NERVE CONDUCTION STUDY OF LOWER EXTREMITIES IN FOOTBALL PLAYERS

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY,

CHENNAI – 600032

In partial fulfillment of the requirement for the degree of

Doctor of Medicine in Physiology (Branch V)

M.D. (PHYSIOLOGY)

MAY 2018

DEPARTMENT OF PHYSIOLOGY

COIMBATORE MEDICAL COLLEGE

COIMBATORE – 14.

CERTIFICATE

This dissertation entitled **"NERVE CONDUCTION STUDY OF LOWER EXTREMITIES IN FOOTBALL PLAYERS"** is submitted to The Tamilnadu Dr. M.G. R Medical University, Chennai, in partial fulfillment of regulations for the award of M.D. Degree in Physiology in the examinations to be held during May 2018.

This dissertation is a record of fresh work done by the candidate **Dr.G.GOWTHAMAN,** during the course of the study (2015-2018). This work was carried out by the candidate himself under my supervision.

GUIDE:
Dr. R. SHANMUGHA VADIVU, M.D.,
Professor,
Department of Physiology,
Coimbatore medical college,
Coimbatore – 14.

PROFESSOR & HOD:
Dr.N.NEELAMBIKAI. M.D.,
Department of Physiology,
Coimbatore Medical College,
Coimbatore – 14.

DEAN:
Dr.B.ASOKAN MS., M.Ch.,
Coimbatore Medical College and Hospital,
Coimbatore – 14.

DECLARATION

I **Dr.G. Gowthaman** solemnly declare that the dissertation entitled "**NERVE CONDUCTION STUDY OF LOWER EXTREMITIES IN FOOTBALL PLAYERS"** was done by me at Coimbatore Medical College & Hospital, Coimbatore, during the period from September 2016 to March 2017 under the guidance and supervision of **Dr.R.Shanmugha Vadivu, M.D.,** Professor, Department of Physiology, Coimbatore Medical College, Coimbatore. This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch - V) in Physiology.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

Place:

Date: **Dr. G. Gowthaman**

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DR. G. GOWTHAMAN

Course : M. D. PHYSIOLOGY

Period of Study : ONE YEAR

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : NERVE CONDUCTION STUDIES OF LOWER EXTREMITIES IN FOOTBALL PLAYERS

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not~~ ~~accepted~~ and you are permitted / ~~Not~~ ~~permitted~~ to proceed with the above Study.

Member Secretary
Ethics Committee

17.3.2016

ACKNOWLEDGEMENT

ACKNOWLEDGEMENT

I express my sincere thanks to our respected Dean, **Dr.B.Asokan, M.S.,M.Ch.,** Coimbatore Medical College, Coimbatore for permitting me to conduct the study.

I thank **Dr.A.Arjunan, M.D.,** Vice Principal, Coimbatore Medical College, Coimbatore for his encouragement and suggestions in completing the study.

I am extremely grateful to my beloved and respected Head of the Department of Physiology, Professor **Dr. N.Neelambikai, M.D.,** for her encouragement in helping me to take up this study. I express my heart - felt gratitude to her, for her moral support and encouragement throughout the conduct of the study and also during my post graduate course. I owe my sincere thanks to her.

I will ever remain in gratitude to **Dr. R. Shanmughavadivu, M.D.,** Professor, Department of Physiology for her valuable support and guidance for my study. I express my gratitude to her for sparing her valuable time and patience, which helped me a lot to complete this study under her expert guidance.

I am highly obliged to **Dr. D.Selvam M.D., DCH,** Associate Professor, Department of Physiology, for his motivation to perform this work.

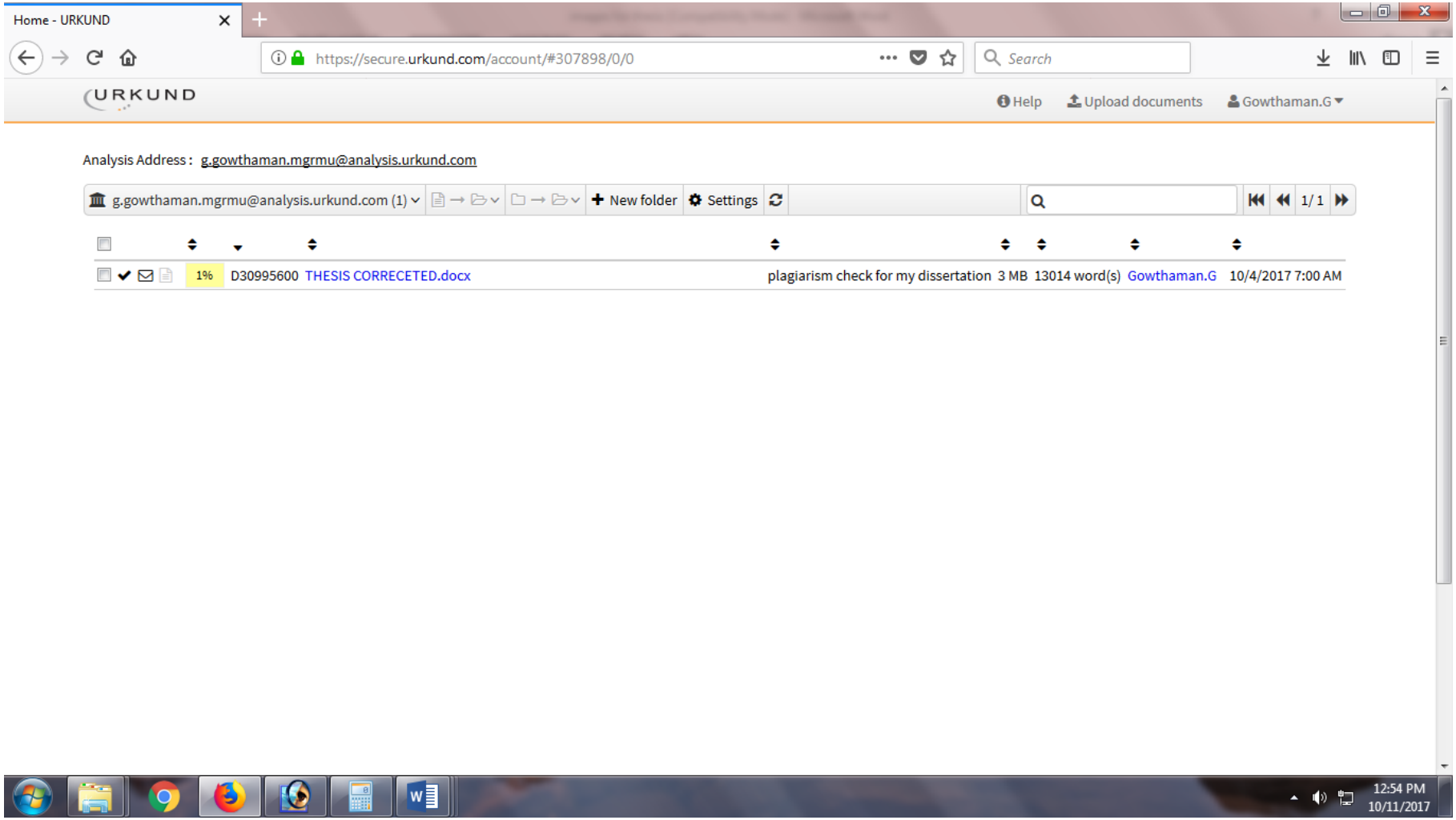
I sincerely thank **Dr. B. Sujatha, M.D., D.A.,** Associate Professor, Department of Physiology for her valuable suggestions and encouragement throughout the period of my study.

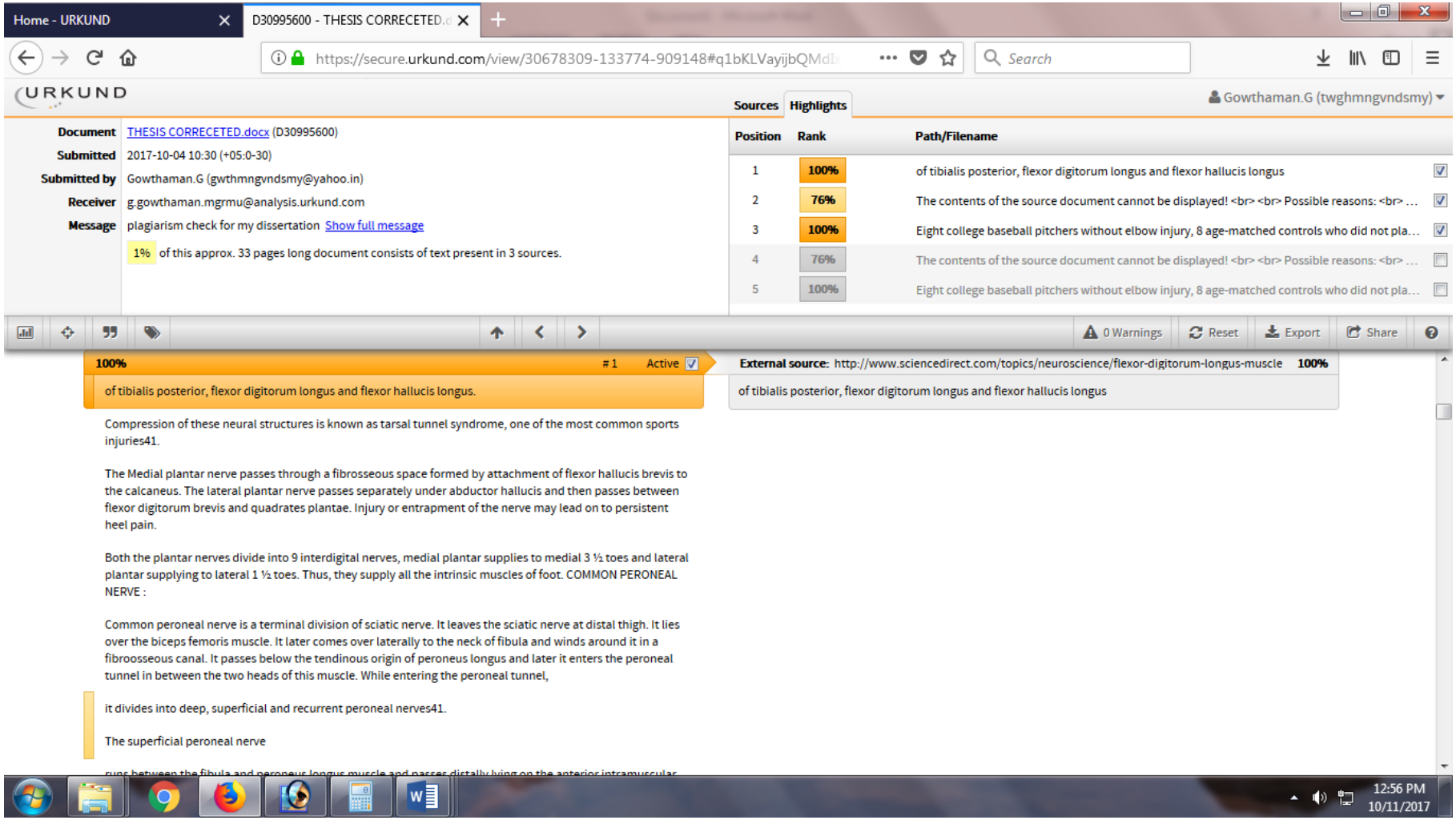
My sincere thanks to beloved teachers **Dr. A. Moorthy M.D., Dr.S.Kavitha M.D., Dr. E.S.Manikandan M.D., Dr.Kanchana Bobby,M.D., Dr. S. Subhashini M.D., Dr. A. Abbas, M.D., Dr. S. Thenmozhi, M.D., Dr.C.N. Angel Deepa, M.D., Mrs. D. Revathy M.Sc.,** Assistant Professors, Department of Physiology for their valuable opinion and help to complete this study. I would like to thank all my **tutors** for their support in completing this study.

I would grossly fail in my duty, if I do not mention here of my **subjects** who gave full cooperation while doing my study.

My sincere thanks to all my **fellow postgraduates** for their involvement in helping me in this work.

My **family and friends** have stood by me, during my times of need. Above all I thank the **Lord Almighty** for His kindness and benevolence


Home - URKUND
https://secure.urkund.com/account/#307898/0/0
Search
URKUND
Help
Upload documents
Gowthaman.G
Analysis Address : g.gowthaman.mgrmu@analysis.urkund.com
g.gowthaman.mgrmu@analysis.urkund.com (1)
New folder
Settings
1/1
1%
D30995600
THESIS CORRECETED.docx
plagiarism check for my dissertation
3 MB
13014 word(s)
Gowthaman.G
10/4/2017 7:00 AM
12:54 PM
10/11/2017


Home - URKUND
D30995600 - THESIS CORRECTED.d
https://secure.urkund.com/view/30678309-133774-909148#q1bKLVayijbQMdb
Search
URKUND
Gowthaman.G (twghmngvndsmy)
Sources
Highlights
Document THESIS CORRECTED.docx (D30995600)
Submitted 2017-10-04 10:30 (+05:0-30)
Submitted by Gowthaman.G (gwthmngvndsmy@yahoo.in)
Receiver g.gowthaman.mgrmu@analysis.urkund.com
Message plagiarism check for my dissertation Show full message
1% of this approx. 33 pages long document consists of text present in 3 sources.
Position Rank Path/Filename
1 100% of tibialis posterior, flexor digitorum longus and flexor hallucis longus
2 76% The contents of the source document cannot be displayed!

 Possible reasons:
 …
3 100% Eight college baseball pitchers without elbow injury, 8 age-matched controls who did not pla…
4 76% The contents of the source document cannot be displayed!

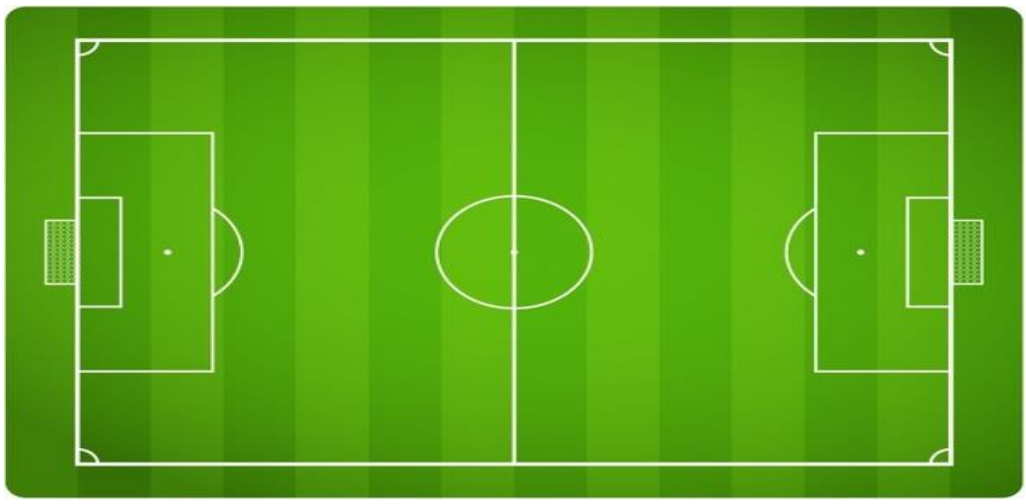
 Possible reasons:
 …
5 100% Eight college baseball pitchers without elbow injury, 8 age-matched controls who did not pla…
0 Warnings
Reset
Export
Share
100% # 1 Active
of tibialis posterior, flexor digitorum longus and flexor hallucis longus.
Compression of these neural structures is known as tarsal tunnel syndrome, one of the most common sports injuries41.
The Medial plantar nerve passes through a fibrosseous space formed by attachment of flexor hallucis brevis to the calcaneus. The lateral plantar nerve passes separately under abductor hallucis and then passes between flexor digitorum brevis and quadrates plantae. Injury or entrapment of the nerve may lead on to persistent heel pain.
Both the plantar nerves divide into 9 interdigital nerves, medial plantar supplies to medial 3 ½ toes and lateral plantar supplying to lateral 1 ½ toes. Thus, they supply all the intrinsic muscles of foot. COMMON PERONEAL NERVE :
Common peroneal nerve is a terminal division of sciatic nerve. It leaves the sciatic nerve at distal thigh. It lies over the biceps femoris muscle. It later comes over laterally to the neck of fibula and winds around it in a fibroosseous canal. It passes below the tendinous origin of peroneus longus and later it enters the peroneal tunnel in between the two heads of this muscle. While entering the peroneal tunnel,
it divides into deep, superficial and recurrent peroneal nerves41.
The superficial peroneal nerve
External source: http://www.sciencedirect.com/topics/neuroscience/flexor-digitorum-longus-muscle 100%
of tibialis posterior, flexor digitorum longus and flexor hallucis longus
12:56 PM
10/11/2017

CERTIFICATE - II

This is to certify that this dissertation work titled **"NERVE CONDUCTION STUDY OF LOWER EXTREMITIES IN FOOTBALL PLAYERS"** of the candidate **Dr. G. Gowthaman** with registration Number **201515252** for the award of **Doctor of Medicine** in the branch of **Physiology**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **1%** percentage of plagiarism in the dissertation.

Guide sign with Seal.

NERVE CONDUCTION STUDY OF LOWER EXTREMITIES IN FOOTBALL PLAYERS



CONTENTS

ABBREVIATIONS

NCS-Nerve conduction study

NCV-Nerve conduction velocity

EMG-Electromyography

CPN-Common peroneal nerve

TTS-Tarsal tunnel syndrome

MRI-Magnetic resonance imaging

TDAL1-Tibial nerve-dominant leg-ankle to foot segment-proximal latency

TDAL2- Tibial nerve- dominant leg- ankle to foot segment-distal latency

TDAA- Tibial nerve- dominant leg -ankle to foot segment-amplitude

TDAD- Tibial nerve- dominant leg -ankle to foot segment-distance

TDACV- Tibial nerve- dominant leg -ankle to foot segment-conduction velocity

TDAF - Tibial nerve- dominant leg -ankle to foot segment-f wave response

TDPL1- Tibial nerve- dominant leg- knee to ankle segment-proximal latency

TDPL2- Tibial nerve- dominant leg -knee to ankle segment-distal latency

TDPA- Tibial nerve- dominant leg -knee to ankle segment-amplitude

TDPD- Tibial nerve- dominant leg -knee to ankle segment-distance

TDPCV- Tibial nerve- dominant leg -knee to ankle segment-conduction velocity

TDAF- Tibial nerve- dominant leg -knee to ankle segment-f wave latency

TNDAL1- Tibial nerve-non dominant leg-ankle to foot segment-proximal latency

TNDAL2- Tibial nerve-non dominant leg-ankle to foot segment-distal latency

TNDAA- Tibial nerve-non dominant leg-ankle to foot segment-proximal latency

TNDAD- Tibial nerve-non dominant leg-ankle to foot segment-distance

TNDACV- Tibial nerve-non dominant leg-ankle to foot segment-conduction velocity

TNDAF- Tibial nerve-non dominant leg-ankle to foot segment-f wave response

TNDPL1- Tibial nerve-non dominant leg- knee to ankle segment-proximal latency

TNDPL2- Tibial nerve-non dominant leg- knee to ankle segment-distal latency

TNDPA- Tibial nerve-non dominant leg- knee to ankle segment-amplitude

TNDPD- Tibial nerve- non dominant leg- knee to ankle segment-distance

TNDPCV- Tibial nerve- non dominant leg- knee to ankle segment-conduction velocity

TNDAF- Common peroneal nerve- non dominant leg- knee to ankle segment-f wave response

CDAL1- Common peroneal nerve -dominant leg-ankle to foot segment-proximal latency

CDAL2- Common peroneal nerve - dominant leg- ankle to foot segment-distal latency

CDAA- Common peroneal nerve - dominant leg -ankle to foot segment-amplitude

CDAD- Common peroneal nerve -dominant leg -ankle to foot segment-distance

CDACV- Common peroneal nerve - dominant leg -ankle to foot segment-conduction velocity

CDAF - Common peroneal nerve - dominant leg -ankle to foot segment-f wave response

CDKL1- Common peroneal nerve - dominant leg- knee to ankle segment-proximal latency

CDKL2- Common peroneal nerve - dominant leg -knee to ankle segment-distal latency

CDKA- Common peroneal nerve - dominant leg -knee to ankle segment-amplitude

CDKD Common peroneal nerve - dominant leg -knee to ankle segment-distance

CDKCV- Common peroneal nerve - dominant leg -knee to ankle segment-conduction velocity

CDAF- Common peroneal nerve - dominant leg -knee to ankle segment-f wave latency

CNDAL1- Common peroneal nerve -non dominant leg-ankle to foot segment-proximal latency

CNDAL2- Common peroneal nerve -non dominant leg-ankle to foot segment-distal latency

CNDAA- Common peroneal nerve -non dominant leg-ankle to foot segment-proximal latency

CNDAD- Common peroneal nerve -non dominant leg-ankle to foot segment-distance

CNDACV- Common peroneal nerve -non dominant leg-ankle to foot segment-conduction velocity

CNDAF- Common peroneal nerve -non dominant leg-ankle to foot segment-f wave response

CNDKL1- Common peroneal nerve -non dominant leg- knee to ankle segment-proximal latency

CNDKL2- Common peroneal nerve -non dominant leg- knee to ankle segment-distal latency

CNDKA- Common peroneal nerve -non dominant leg- knee to ankle segment-amplitude

CNDKD- Common peroneal nerve - non dominant leg- knee to ankle segment-distance

CNDKCV- Common peroneal nerve - non dominant leg- knee to ankle segment-conduction velocity

CNDKF- Common peroneal nerve - non dominant leg- knee to ankle segment-f wave response

INTRODUCTION

INTRODUCTION

The game of football is both an art and science. It involves techniques like running, kicking, passing, heading, juggling, dribbling etc. These activities are often performed at great speed and aggression. The game of football contains a lot of physical challenges. It is a game of constant action.

In football, the body size, shape and composition of players play a significant role. There is a strong correlation between anthropometric measurements and success in football. The game includes sprint and jump in both attack and defense for which anthropometric measurements play a crucial role[1].

The game is played for 90 minutes. For the whole time, the entire body weight and stress of playing is concentrated on the lower limbs alone. This induces both physiological and pathological changes in the physique of the individual. It includes changes in the muscles, tendons, bones, soft tissues, joints etc[2].

The physiological profile of the players play a vital role in selection of athletes for the team. Hence they cannot be permitted to have the freedom of injuries. For this, the players undergo extensive cumbersome training routines. On occasions, even these training routines pose a threat of serious injuries to players[3]. This study focuses on the amount of wear and tear the sport has on the lower limb nerves of the football players.

Football is sometimes dangerous leading to injuries like complicated fractures, joint dislocations, muscle tear etc. Successful performance of the athletes depends upon the anthropometric morphological characteristics[1], physical ability, explosive power, anaerobic and aerobic capacity. To achieve this, the players are subjected to enormous amount of training which takes a toll on their body, especially nerves.

The activities carried out in football are multi directional, intermittent, varying in intensity and time. There is sudden acceleration and deceleration of the body in the direction of motion which challenges the players. It is important for footballers to have isometric strength which they acquire over long periods of training.

The endurance level which the footballers show is maximum and they achieve it at the cost of their physical well being. This study focuses on the effect of football and its training on the lower limb motor nerves of the players.

The effect of football on lower limb nerves of the individual is checked through nerve conduction study. It is an electrodiagnostic procedure in which small amount of current is given to an electrode placed over the nerve and the response is obtained through another electrode placed over the muscle. In this study the nerve conduction studies of tibial nerve and common peroneal nerve are done.

AIM & OBJECTIVES

AIM & OBJECTIVES

AIM :

The aim of the study is to determine the nerve conduction parameters like latency, amplitude, conduction velocity and F – wave response of football players and comparing with normal controls.

OBJECTIVES :

1. Comparison of motor nerve conduction parameters like latency, amplitude, conduction velocity and f-wave response of football players and normal controls.

2. Analysis of motor nerve conduction parameters like latency, amplitude, conduction velocity and F-wave response of football players and normal controls.

HISTORY

HISTORY

The early development of nerve conduction studies was connected to the discovery of electricity. This was concluded so because the nerves and muscles themselves produced electricity when electric current was applied to the body of the animal.

The main contributors for the development of nerve conduction studies were

Galvani

Gasser and Erlanger

Matthews

Adrian and Bronk

Denny brown & Penny backer

Larrabee, Hodes and German

Lambert and Eaton

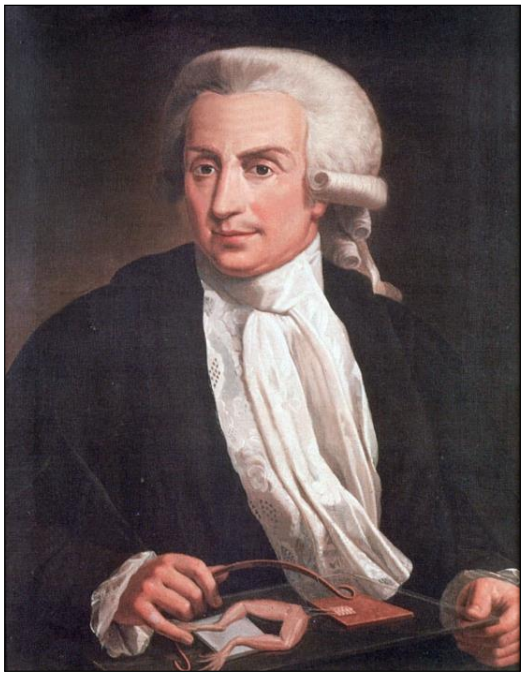
The research in this topic was heralded by physiologists then carried forward by neurologists.

World War II played a mixed role in the evolution of research and technology. It was during the time of world war various researches were conducted on the injured soldiers.

Galvani

Luigi Aloissio Galvani born in 9 September 1737 was an Italian Physician Physicist who discovered animal electricity. He was a pioneer in

LUIGI GALVANI



bioelectromagnetics. He found out that when muscles of frog were struck by electric current it produced twitches in them[4].

The beginning of Galvani's experiment with bioelectricity has a popular legend which says that Galvani was slowly skinning a frog at a table where he had been conducting experiments with static electricity by rubbing frog skin[5].

Galvani's assistant touched the exposed sciatic nerve of frog with a metal scalpel that had picked up a charge. Then later dead frog's legs kicked as if in life. Thus Galvani was the first investigator to appreciate relationship between electricity and life. This proves to be the first and foremost work in relation to the nerve conduction studies[6].

He proved that the movement of ions within the body is responsible for muscular twitches which was contrary to balloonist theories. The balloonist theory suggested that the movement of air or fluid into the muscle was responsible for those twitches. This theory was put forward by Galen. Later it was disproved by Jan Swammerdam[6].

Galvani coined the term animal electricity to describe the effects of electric current on animal muscles. It was later termed 'Galvanism' by the physicist Volta. Galvani was properly credited with the discovery of bioelectricity Today, the study of galvanic effects in biology is called electrophysiology. Galvani died in Bologna on 4 December 1798[7].

HERBERT SPENCER GASSER

JOSEPH ERLANGER

Herbert Spencer Gasser:

Gasser was born on July 5, 1888 in Wisconsin, U.S.. He was an American physiologist. He studied physiology under Joseph Erlanger. He graduated in medicine from John Hopkins university in 1915[8,9]. In 1936 Gasser along with Erlanger gave a series of lectures summarizing their work into the actions of human nerve cells, at the university of Pennsylvania. This work led to recognition in 1944, when they jointly received Nobel prize[10,11]. He died in New York on May 11, 1963.

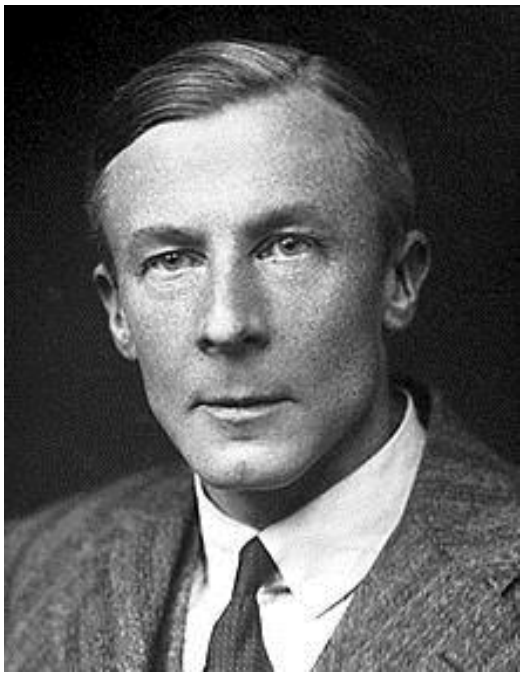
Joseph Erlanger :

He was an American physiologist. He was born in Jan.5.1874 at San Francisco, California. He completed his MD in 1899 from John Hopkins University. He worked under William Osler. He was interested in Cardiology and developed and patented a new type of sphygmomanometer which measured B.P. from brachial artery[12].

Erlanger and Gasser were able to modify a western electric oscilloscope to run at low voltages. Using this, they were able to observe the action potentials. They also discovered that the velocity of action potentials was directly proportional to the diameter of nerve fiber. In 1944, they won Noble prize in Medicine or physiology for these discoveries[11]. He died in Dec.5 1965 at St. Louis, Missouri.

BRYAN MATHEWS

EDGAR ADRIAN



DETLEV BRONK

BRYAN MATHEWS :

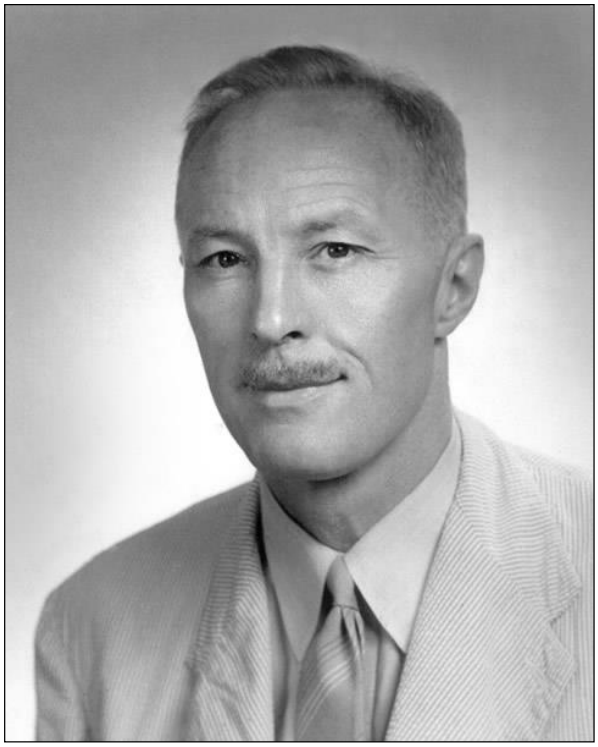
Sir Bryan Harold Cabot Mathews born in 14th June 1906 was a Professor of Physiology at Cambridge University from 1952 – 1973. He was a student of Adrian. He designed moving iron oscillograph which was immediately adopted by Adrian and others for photographing the traces of single nerve impulses in the spate of experiments that followed the first single unit records of 1925. He also developed a differential amplifier used by all electrophysiologists. He was engaged investigating the single nerve fibre responses of muscle spindles first in frog and then in cat[13].

Mathews with Adrian as a subject demonstrated the Berger waves using his amplifier at Cambridge meeting of physiological society in 1934 which launched the science of EEG. For his achievements he was elected as FRS in 1940[14]. He died in 22 July 1986 aged 80.

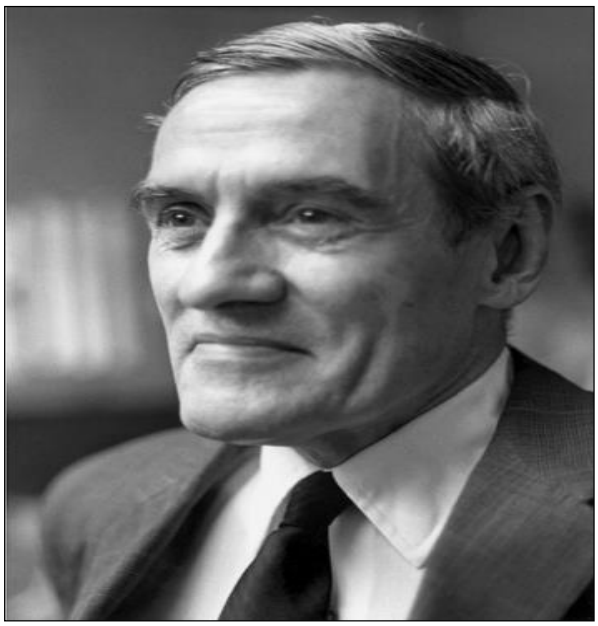
ADRIAN AND BRONK :

In 1929 Adrian and Bronk demonstrated a more refined method of obtaining action potentials not only in animals but also in man. They inserted small concentric electrodes into the single motor units and obtained action potentials. The electrodes are made of fine insulated copper wire of 36μ diameter inserted into a 25 gauge steel hypodermic needle. In 1932 Adrian won Nobel prize for physiology along with Sherrington[15,16]. Bronk was an American biophysist. In 1926 he got his

DENNY BROWN



LARRABEE



PhD from the university of Michigan for applying physics and mathematics to physiology[17].

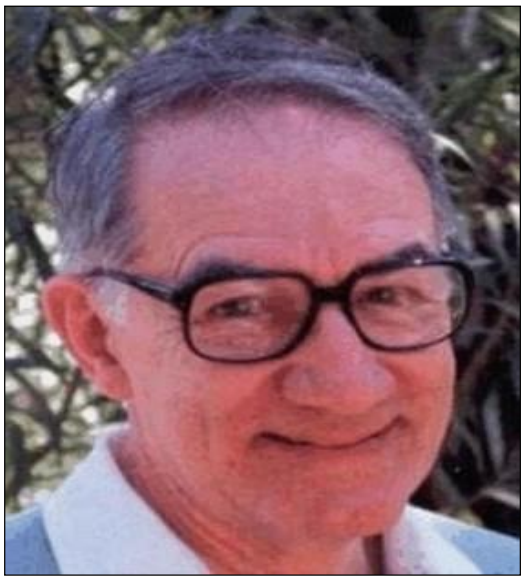
DENNY BROWN :

Derek earnest Denny-Brown was a New Zealand born neurologist. He did pioneering work in development of electromyography as an electrodiagnostic technique[18]. He qualified in medicine from the university of Otago in 1924. He did his fellowship under Dr. Sir Charles Scott Sherrington. In 1928 he moved to London to become a neurologist. He moved to US in 1941 assuming the directorship of neurology at Boston City Hospital and gained US citizenship in 1952. In 1981 he died from multiple myeloma. It was in 1938 he laid foundation for clinical electromyography with Pennybacker[18]. They recorded the action potentials of single contracting or spontaneously firing motor units called fasciculations and were separated from action potentials of single denervated motor fibres called fibrillations[19].

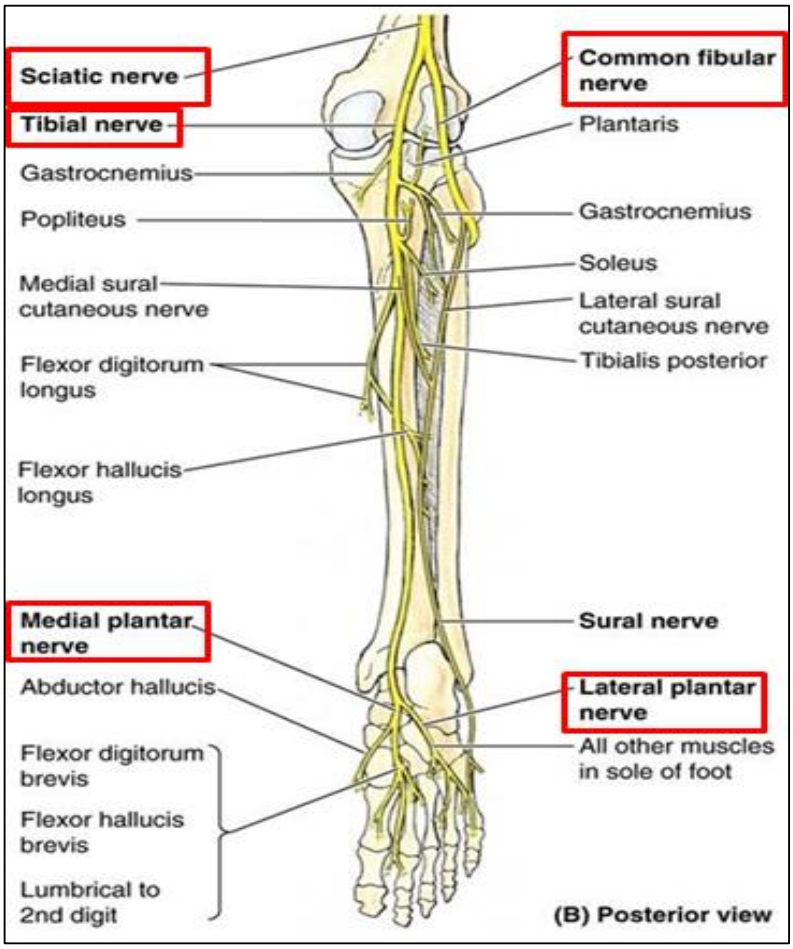
LARRABEE :

Martin Larrabee was an American biophysicist born in Masscheusetts. He along with Hodes and German recorded the compound muscle action potential in healthy and injured nerves of world war II victims[20].

Dr. LAMBERT



ANATOMY OF TIBIAL NERVE



LAMBERT AND EATON

In 1957, Lambert and Eaton described the electro physiologic features of a new myasthenic syndrome associated with lung carcinoma[21]

Thus, the research process of nerve conduction studies was pioneered by physiologists and carried forward by neurologists[21].

ANATOMY:

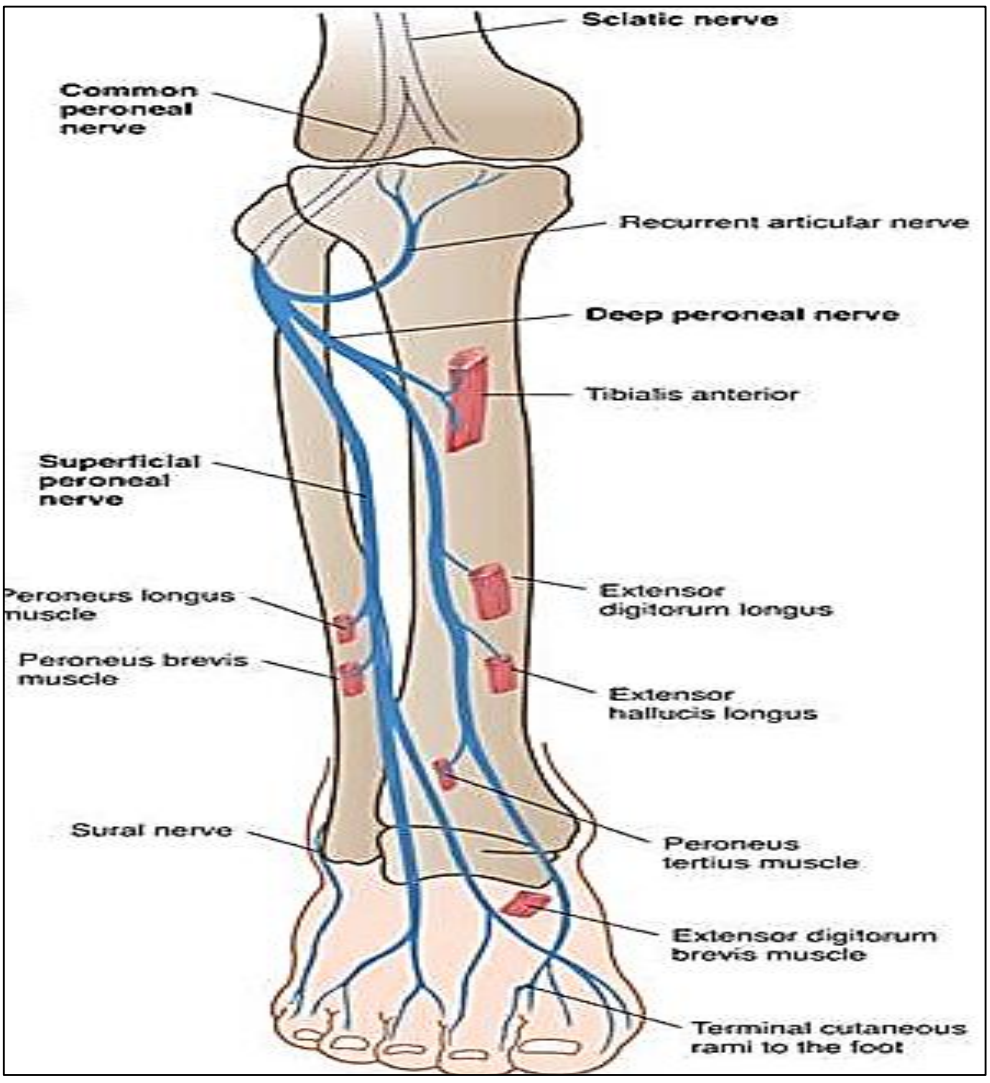
TIBIAL NERVE :

Tibial Nerve is a branch of sciatic Nerve. It originates from the anterior divisions of $L_4 - S_3$ via the medial trunk of sciatic nerve. It arises in the popliteal fossa where sciatic nerve divides into tibial and common peroneal nerves[22].

It supplies gastrocnemius, soleus, tibialis posterior, flexor digitorum longus and flexor hallucis longus in the calf. It becomes superficial about 15cm above the ankle medial to achilles tendon. Then it undermines the flexor retinaculum thus forming the root of tarsal tunnel. Within the tunnel it divides into medial and lateral plantar nerves supplying the foot. Compression at this tarsal tunnel is called tarsal tunnel syndrome.

The tarsal tunnel is bridged by the retinaculum and medial border of tunnel is talus and calcaneus. The tibial nerve and its branches pass through distinct tunnels separate from posterior tibial vessels and tendons of tibialis posterior, flexor digitorum longus and flexor hallucis longus.

ANATOMY OF COMMON PERONEAL NERVE



Compression of these neural structures is known as tarsal tunnel syndrome, one of the most common sports injuries[22].

The Medial plantar nerve passes through a fibrosseous space formed by attachment of flexor hallucis brevis to the calcaneus. The lateral plantar nerve passes separately under abductor hallucis and then passes between flexor digitorum brevis and quadrates plantae. Injury or entrapment of the nerve may lead on to persistent heel pain[23].

Both the plantar nerves divide into 9 interdigital nerves, medial plantar supplies to medial 3 ½ toes and lateral plantar supplying to lateral 1 ½ toes. Thus, they supply all the intrinsic muscles of foot.

COMMON PERONEAL NERVE :

Common peroneal nerve is a terminal division of sciatic nerve. It leaves the sciatic nerve at distal thigh. It lies over the biceps femoris muscle. It later comes over laterally to the neck of fibula and winds around it in a fibroosseous canal[24]. It passes below the tendinous origin of peroneus longus and later it enters the peroneal tunnel in between the two heads of this muscle. While entering the peroneal tunnel, it divides into deep, superficial and recurrent peroneal nerves[22].

The superficial peroneal nerve runs between the fibula and peroneus longus muscle and passes distally lying on the anterior intramuscular septum. It supplies both the peroneal muscles. It pierces the crura fascia at the junction of middle and distal third of tibia and splits into two

cutaneous branches. It is here where the nerve may be compressed at the crura fascia. It mainly occurs due to thickening of crura fascia known as superficial peroneal nerve syndrome[22,25,26].

The deep peroneal nerve pierces the anterior intermuscular septum and accompanies anterior tibial blood vessels. There it lies between tibialis anterior and extensor hallucis longus distally. It also innervates extensor digitorum longus and peroneus tertius.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

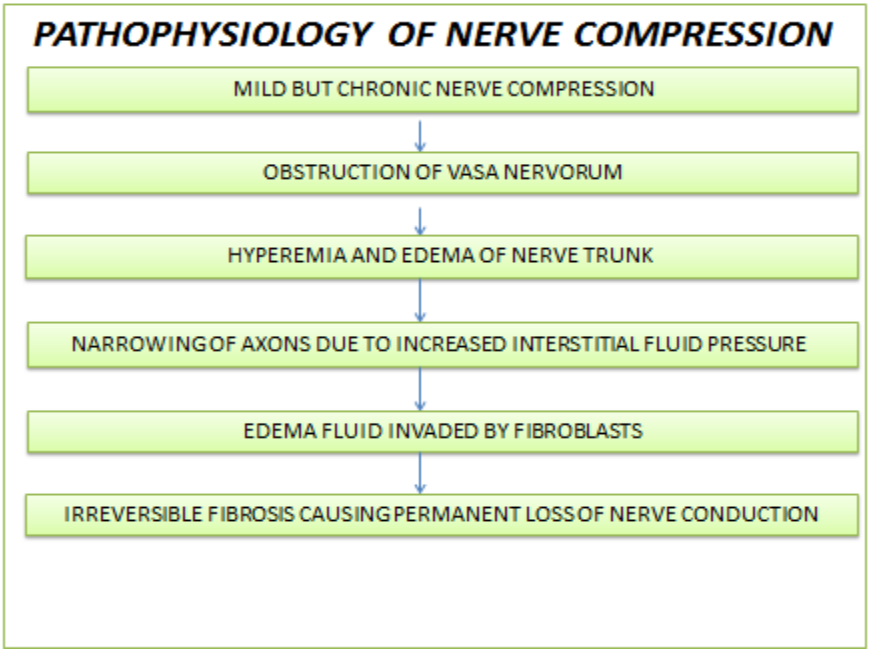
ETIOLOGY AND CLINICAL PRESENTATION OF INJURIES

Athletes who are involved in sports are exposed to certain injuries specific to a particular sport including injuries to foot and ankle. Approximately 25% of sports injuries involved foot and ankle of which 45% are lateral ankle sprains[27].

Most of the sports especially football poses a specific threat to athlete's foot as a result of activity and equipment that is used. Most of the injuries are not benign. Approximately 40% of all simple ankle sprains lead to chronic instabilities[27].

The etiology, clinical presentation of various lower limb football injuries and how they lead onto changes in nerve conduction parameters is as follows.

PATHOPHYSIOLOGY OF NERVE CONDUCTION[28,24]:



1. **Ankle instability :**

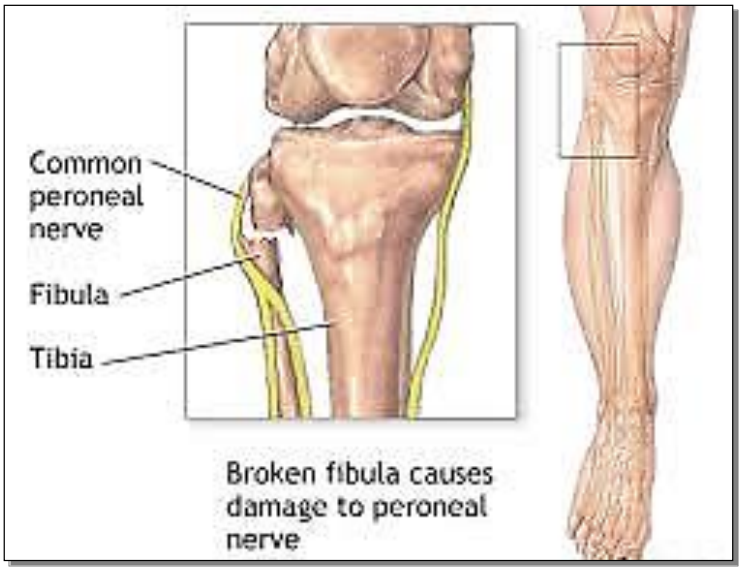
It is one of the common injuries sustained in football. The recurrence rate after an ankle sprain is high. It occurs because of an excessive supination of foot in a laterally rotated leg. The excessive supination occurs at subtalar joint. Typically it presents with an audible POP or a click. Later ankle gets swollen, tender and painful while doing joint movement and weight bearing. It is identified by the tenderness at the level of lateral malleolus and when trying to evert the foot[29]. It is also associated with injury to tibiofibular syndesmosis which is tested by squeezing the lower end of tibia and fibula together. It is appreciated by increased tenderness at the site. This test is called Hoplainson's syndesmotic squeeze test[27].

Complications :

Lateral ankle sprain might lead onto alterations in the alignment of subtalar joint. It is commonly associated with compression of tibial nerve as it passes below the subtalar joint which may lead on to changes in the nerve conduction parameters[30,31].

X-rays sometimes shows a fibular fracture, anterior process of calcaneus fracture, midtarsal fracture or disruption of ankle mortice. This disruption of the ankle mortice is the prime cause of injury to the underlying nerves[32,33].

COMMON PERONEAL NERVE INJURY



2. Common Peroneal Nerve Syndrome :

It is diagnosed by increased pain while running or doing exercise. In case of injury to CPN, there will be weakness of muscle, paraesthesia and positive tinel sign at fibular neck level[34]. Care should be taken to rule out anterior compartment syndrome which also presents with pain related to exercise. It is characterized by pain along the distribution of nerve whereas anterior compartment syndrome is associated with generalized pain.

It is of two types

Superficial Peroneal Nerve Syndrome

Deep Peroneal Nerve Syndrome

Superficial Peroneal Nerve Syndrome :

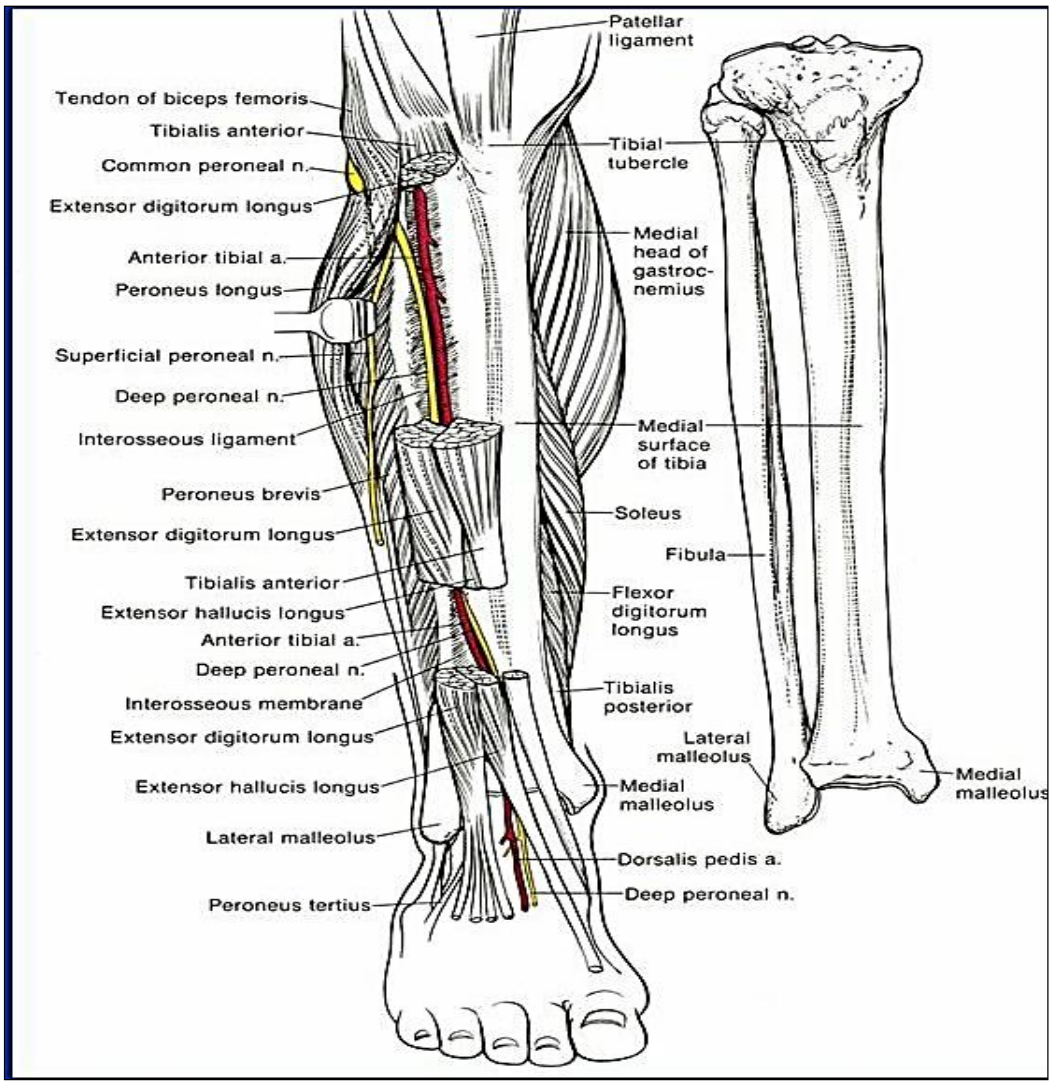
It is associated with pain and paraesthesia along the lateral calf[34]. There is difficulty in doing dorsiflexion and eversion of foot. It is more commonly found in soccer players[22,25].

Deep Peroneal Nerve Syndrome :

It is more commonly associated with pain and paraesthesia along the dorsum of foot. Its clinical features are similar to Tarsal tunnel syndrome.

The major problem underlying the CPN compression at the level of fibular neck is the presence of a ganglion cyst compressing it[26]. It is relieved by surgical excision of the ganglion cyst. Thus, the nerve is

COMMON PERONEAL NERVE SYNDROME



decompressed. It is usually diagnosed by Electromyography and nerve conduction studies. NCS usually demonstrates a slowing of nerve conduction at the peroneal tunnel.

The compression of CPN at the level of fibular neck is the most common compression neuropathy involved in sports with regard to lower limbs[22,35]. It is due to the pressure from the overlying muscle. The nerve travels beneath the sharp fibrous origin of peroneus longus which accounts for the increased risk of compression[33].

It is treated by oblique incision along the course of the nerve exposing the nerve and fibrous origin of peroneus longus muscle. The fascia overlying the muscle is also divided. This decompresses the nerve and the patient is relieved of the symptoms in 2-3 weeks[36].

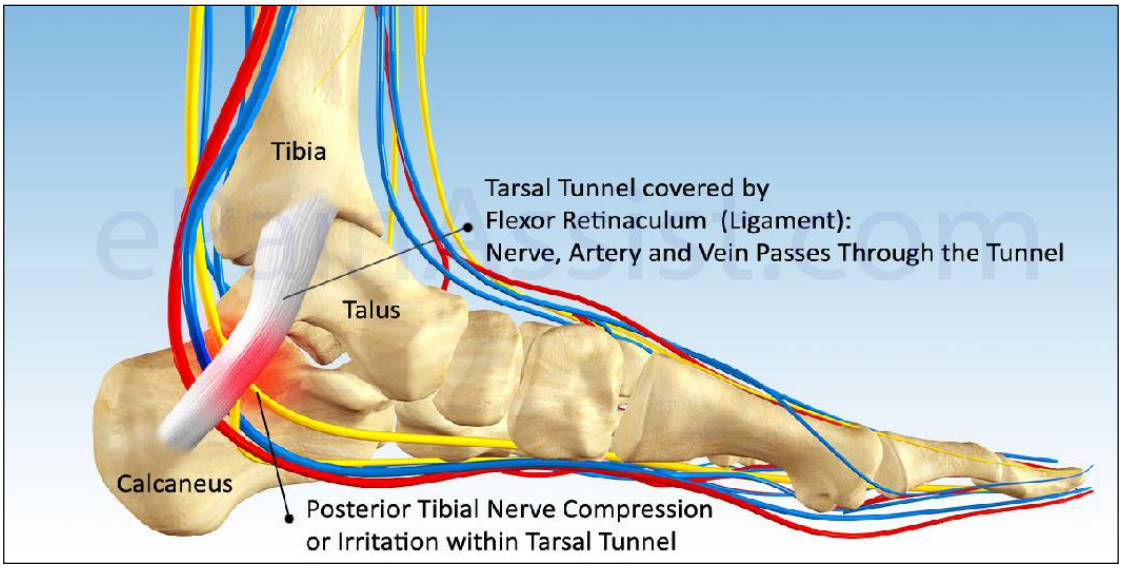
3. TIBIAL NERVE :

TARSAL TUNNEL SYNDROME(TTS):

It occurs because of the damage to tibial nerve or its branches within the tarsal tunnel. It leads to pain and paraesthesia along the dorsum of foot. It usually occurs because of alteration in the anatomy of tarsal tunnel such as

1. Ligamentous trauma to ankle
2. Mass lesion
3. Local bony prominence Eg. a spur
4. Local tenoperiostitis[22]

TARSAL TUNNEL SYNDROME



Usually during operations for TTS, Ligamentous thickening idiopathic fibrosis have found to be frequently encountered[37].

It is diagnosed by nerve conduction studies and electromyography. MRI Scan further delineate the tunnel accurately and is confirmatory for TTS.

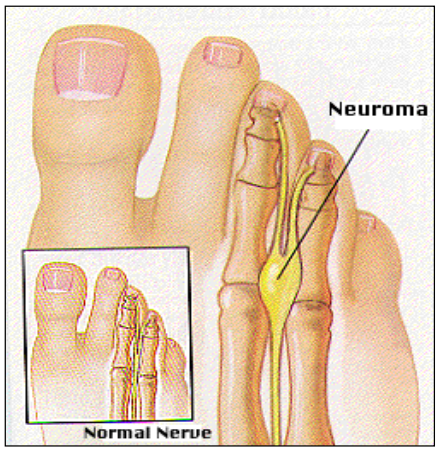
The treatment of tarsal tunnel syndrome is aimed at correction of biomechanics first[22]. That is correction of foot wear, use of orthotics etc. Next is the injection of steroids into the tarsal tunnel to reduce inflammation. Care should be taken to inject the steroids into the tarsal tunnel because of its complex anatomy. The last and the most effective option is surgical exploration of the tarsal tunnel. The athlete can return to sports within a month after the surgery[38].

4. MORTON NEUROMA :

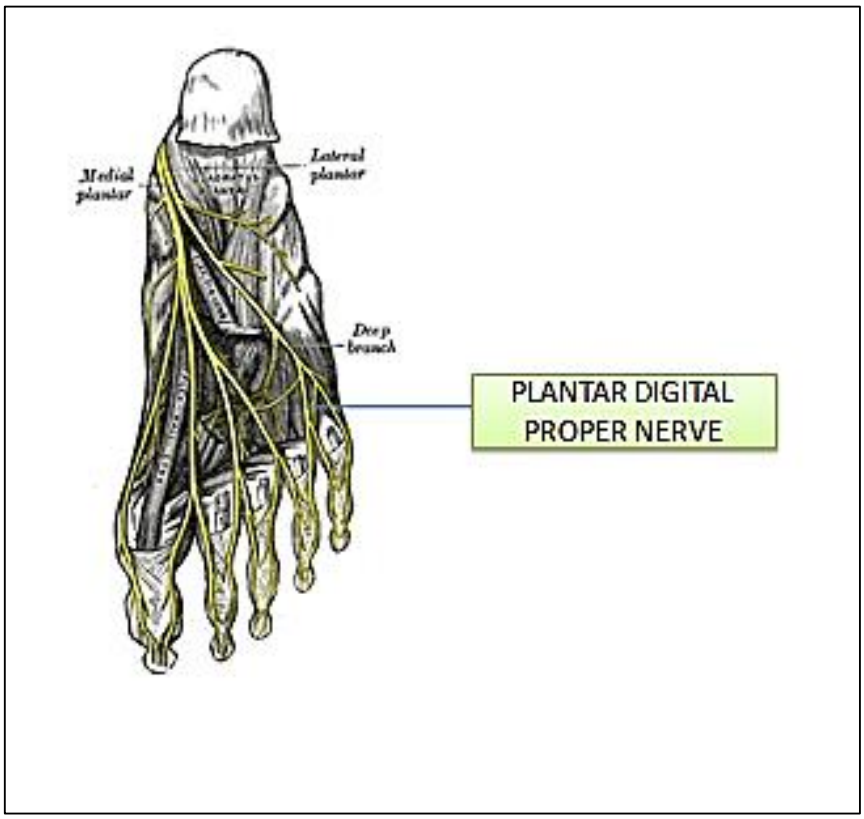
It occurs due to injury to the inter digital nerve at deep transverse metatarsal ligament[39]. It lies between two heads of adjacent metatarsal bones. When compression injury[40] causes approximation of metatarsal heads it leads to compression of the inter digital nerves[22]. This leads to fibrosis of nerves.

The athlete usually presents with fore foot pain radiating to the toes. When metatarsal heads are compressed it leads to shooting pain. Exercise increases pain[39].

MORTON NEUROMA



JOPLIN'S NEURITIS



Electrophysiological tests, MRI confirm the diagnosis[38].

It is treated with foot wear modification, Metatarsal padding and corticosteroid infiltration. If symptoms persist neurolysis can be done[22].

5. JOPLIN'S NEURITIS :

It is caused by the injury to medial plantar proper digital nerve. It occurs due to wearing an inadequate footwear causing chronic compression. It occurs where the nerve crosses the first metatarsophalangeal joint or medial aspect of great toe[41].

Typical presentation is pain and paraesthesia along the medial side of great toe. Symptoms usually begins because of wearing a tight foot wear and then remains as a persistent area of muscle pathologies[22].

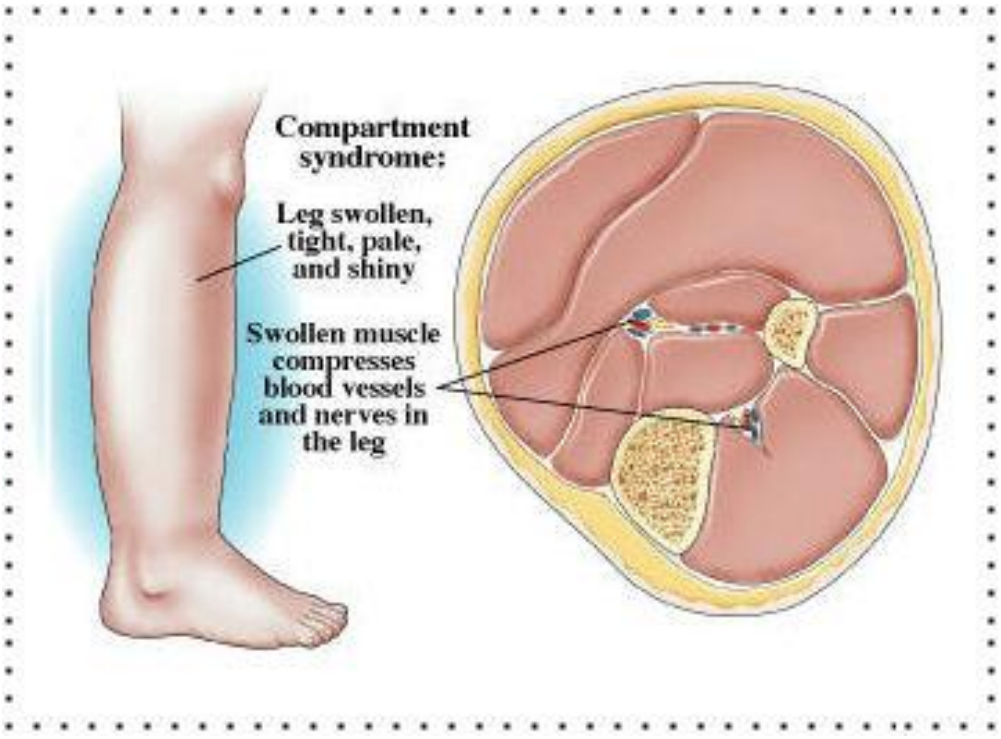
6. **EXERTIONAL COMPARTMENT SYNDROME** :

It is characterized by

- Dull aching pain in the antero lateral compartment of leg.
- Insidious in onset with activity and relieved by proper rest.
- No history of acute trauma
- Pain reproducible after exercise for few minutes[2].

Exertional compartment syndrome occurs because of the compression of calf muscles during a strenuous physical exercise. As and when the athletes exercise, the blood to the muscles of the compartment increases. Muscles increases in volume whereas the fascia which encases

EXERTIONAL COMPARTMENT SYNDROME



the muscles will not expand. This leads to increase in compartmental pressure. This is called exertional compartmental syndrome[42,43,44].

Chronic compartmental syndrome can lead onto scarring of the fascia[27]. This causes compression of the nerves coming out of or piercing through the fascia which leads to neurological symptoms[45].

Typically a compartmental syndrome can be reproduced if the athlete exercises for a particular period of time. Hence it is also called as third lap syndrome.

On examination the affected compartment may be tender and slightly swollen. In few patients, there is herniation of the muscle which can be palpated.

Diagnosis :

Chronic compartmental syndrome shows scarring in an MRI. The calf muscle edema can be noted in an acute exertional compartmental syndrome in a MRI.

The gold standard is measurement of compartmental pressure pre and post exercise.

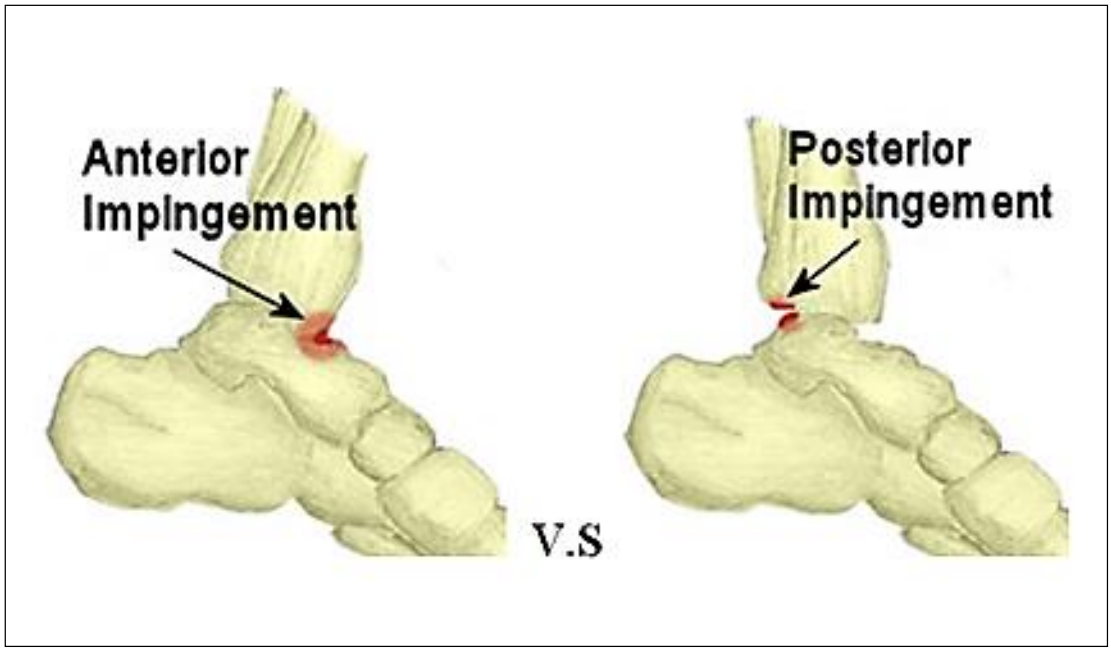
Pre exercise pressure must be equal or higher than 15mm Hg.

One-minute after exercise pressure must be above 50mm Hg.

5- minutes post exercise pressure above 15mm Hg.

If any one of the above said 3 criteria's are met it is diagnosed as compartmental syndrome[46,47].

TIBIO TALAR IMPINGEMENT



Treatment :

NSAIDs may offer some help initially.

If symptoms persist then careful meticulous surgical release of compartment is the treatment of choice. The athlete can return to full exercise program within 8-12 weeks after surgery.

7. ANTERIOR TIBIO TALAR IMPINGEMENT :

It is also called as Footballer's ankle[26]. It is usually seen in athletes exposed to increased dorsiflexion of ankle. This leads to chronic sprains of anterior ankle capsule and microtrauma to anterior cartilage cap of distal tibia. This leads onto subsequent calcification and formation of bony spurs[48].

These bony spurs may impinge upon the nearby nerves which leads onto neurological symptoms. If the bony spurs are large they may be impinge upon themselves limiting the joint movements[48].

The athlete usually presents with history of anterior ankle or midfoot pain radiating towards the lateral aspect of ankle joint or fibula. They also report stiffness of ankle[40].

It is diagnosed by the presence of bony spur on the dorsal surface of the neck of the talus. There is loss of round contour of anterior margin of tibia. Sometimes loose bodies can be seen in the joint space[2].

Treatment is NSAIDs for period of few weeks followed by immobilization cast for 4-6 weeks. If symptoms persists the treatment is surgery. It is usually treated arthroscopically for removal of bony spur and loose bodies.

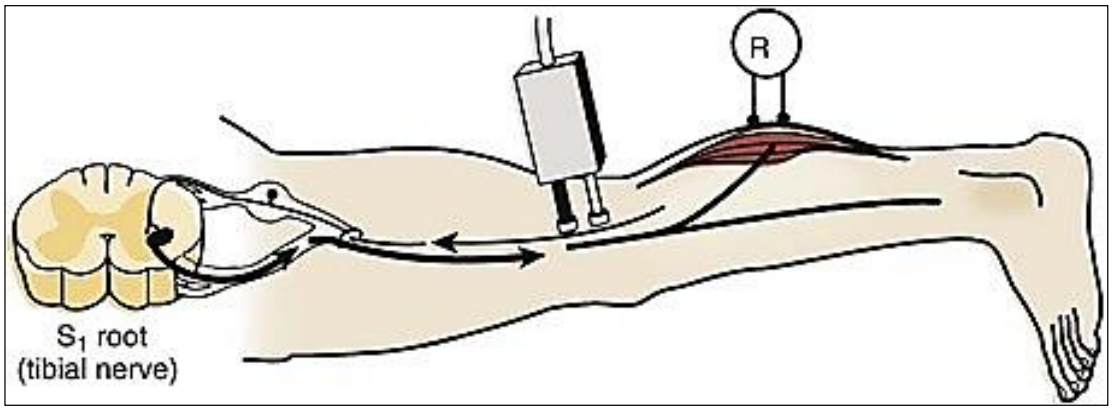
NERVE CONDUCTION STUDY :

It is an electro diagnostic procedure used to study neural pathology. It is very sensitive in detecting nerve entrapment or compression neuropathies and peripheral neuropathies[49]. In this, a nerve is electrically stimulated and the response is obtained from the nerve itself or the muscle which it supplies. It evaluates both the structural and function changes in the nerves which aids in predicting the course of a neural disease.

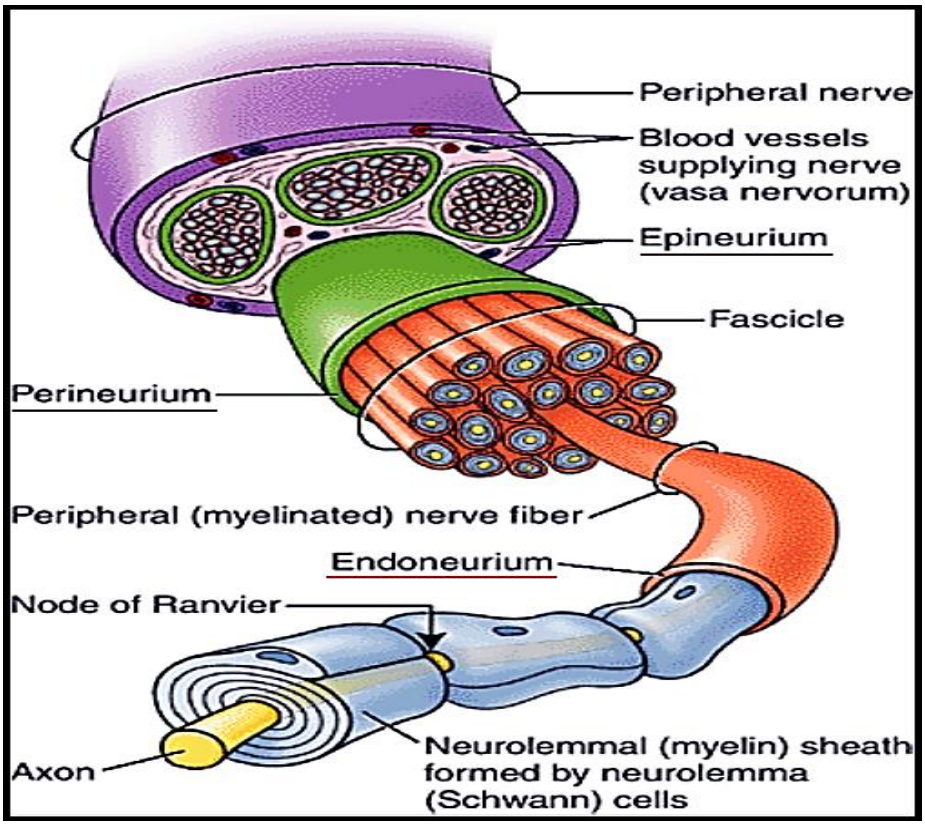
INDICATIONS :

1. To localize the site of level of lesion.
2. To know if the injury is due to axonal loss or demyelination.
3. To diagnose mononeuropathies[50]
4. To diagnose peripheral neuropathies and inflammatory neuropathies Eg.GBS[49,51].

PRINCIPLE OF NERVE CONDUCTION STUDY



ANATOMY OF NERVE



PRINCIPLE :

In this mild electrical shock is applied to one point of the nerve and the signal response is recorded from another point. The other point may be present over the nerve itself or the muscle.

ANATOMY AND PHYSIOLOGY OF NERVE CONDUCTION

ANATOMY:

Peripheral nerves are made up of fascicles. Each fascicle consists of a bundle of nerve fibres. Each bundle of nerve fibres consists of three sheaths of connective tissue namely endoneurium, perineurium and epineurium from inside out[52].

Endoneurium is the connective tissue which surrounds the individual nerve fibres. It is placed longitudinally along the course of the nerve fibres. Endoneurium is present between the surface membranes of Schwann cells in which axons are embedded[53,54].

Perineurium is the connective tissue which surrounds each fascicle. It is made of flat polygonal cells. They form tight junctions which forms a continuous membrane. It forms the blood nerve barrier[55,56]. It also forms the diffusion barrier. It provides tensile strength to the nerves[57].

Epineurium is the connective tissue which surrounds the fascicles. It is loosely bound to the fascicles. It is made of collagen and fat. It contains blood vessels and lymphatics. It continues with duramater of spinal nerve root[58,59].

Peripheral nerves are made up of afferent and efferent nerve fibre. Afferent fibres are sensory and enter into spinal cord via posterior root which convey impulses to brain. Efferent fibres are motor that leave spinal cord via anterior roots and supply the muscles. The afferent and efferent fibres have a central neuron which is located in dorsal root sensory ganglion or anterior horn cell of spinal cord respectively from which axon arises.

CLASSIFICATION OF NERVE FIBRES :

Conduction velocity of nerve depends upon :

1. Fiber diameter
2. Degree of myelination
3. Internodal distance.

Based on fiber diameter, the nerve fibers are classified into 3 types

Group A

Group B

Group C

Group A fibers contain both afferent and efferent myelinated somatic fibres of small, medium and large diameter. They are sub classified into $\alpha,\beta,\gamma,\varepsilon,\eta$ in order of descending diameter and conduction velocity.

Group B fibers consist of only small pre-ganglionic myelinated axons of autonomic nervous system.

Group C fibres consist of unmyelinated fibres which are visceral afferents, Pain & temperature afferents and preganglionic autonomic efferent.

Based on Myelination, the fibers are classified into

Myelinated fibres

Unmyelinated fibres

Myelin sheath is formed by schwann cells. Junction between two schwann cells is called Node of Ranvier. The distance between two nodes of Ranvier is called internodal distance. The nodes of Ranvier alone is unmyelinated from where the action potential originates[24].

ERLANGER & GASSER CLASSIFICATION

Erlanger /Gasser classification of nerve fibers

Fiber types	Function	Avg. fiber diameters (µm)	Avg. cond. Velocity (m/s)
Aα	Primary muscle spindle afferents, motor to skeletal muscle	15	100 (70-120)
Aβ	Cutaneous touch and pressure afferents	8	50 (30-70)
Aγ	motor to muscle spindle	5	20 (15-30)
Aδ	Cutaneous temperature and pain afferents	<3	15 (12-30)
B	Sympathetic preganglionic	3	7 (3-15)
C	Cutaneous pain afferents sympathetic postganglionic	1	1 (02-2)

IMPULSE CONDUCTION :

Action potential originates in the nodes of Ranvier. It can be conducted towards either sides. The conduction is continuous in unmyelinated and saltatory in myelinated fibers.

MYELINATED FIBRES :

Myelination helps in increased conduction velocity of nerves. Thicker the myelin sheath faster is the conduction. Thinner the sheath or if there is demyelination, conduction become slow which is called conduction block. This occurs because of greater loss of local current before reaching the next node of Ranvier. This is usually the scenarios which occurs in case of compressive neuropathies of sports.

UNMYELINATED FIBERS :

Unmyelinated fibres conduct nerve impulses slowly and continuously. In cases of local compression the conduction velocity is further slowed.

PHYSIOLOGICAL ASPECTS OF NERVE CONDUCTION :

Nerve cells gets excited even at a low threshold stimulus. Those stimuli can be electrical, chemical or mechanical. The action potential which arise because of those stimulus forms the main language of the nervous system.

RMP :

There exists an osmotic equilibrium within the nerve cells at rest. This equilibrium is maintained by the concentration of various ions within and outside the cells. The cell membrane is selectively permeable for

those ions. The concentration of sodium ions is more outside the cell and concentration of potassium ions is more inside the cell. This gives rise to a potential difference across the membrane[54].

When the cell is at rest, it is more permeable to potassium ions. All of the sodium channels are closed. However there is some leak of sodium ions inside the cells which is kept in check by the Na^+K^+ ATPase pump. Chloride ions make no contribution. Thus, Resting Membrane potential solely depends on the influx of K^+ions[52].

The equilibrium potential at which there is no movement of ions is explained by Nernst equation.

Nernst equation :

$$E(ion) = \frac{RT}{NF} \frac{Log(Ion)\ outside}{Log(ion)\ inside}$$

Where,

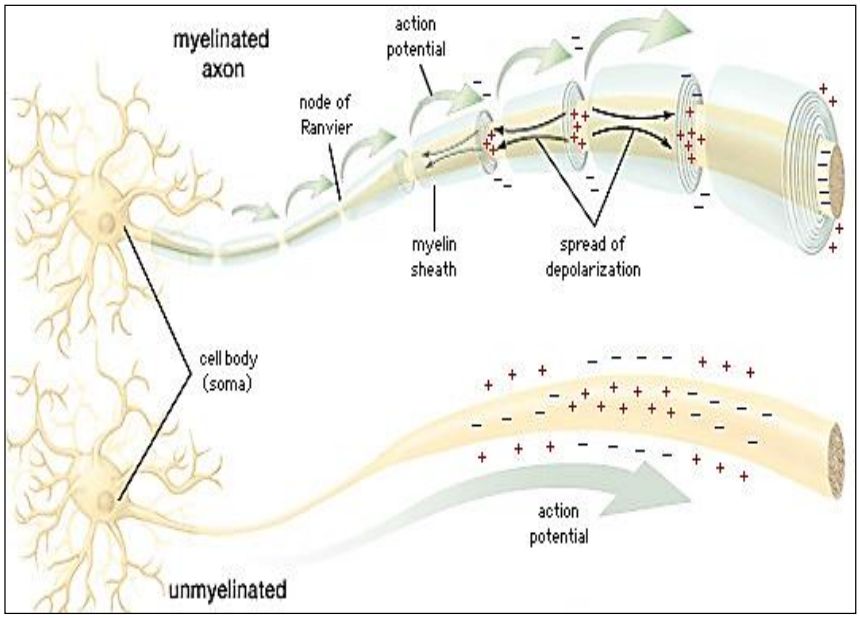
R - Gas constant

F - Faraday constant

N - Valency of ion

T - Temperature

IMPULSE CONDUCTION IN NERVES



ACTION POTENTIAL :

Action potentials arise when the membrane is stimulated by a stimulus above the threshold level. There are two types of action potentials,

Propagated APs

Non propagated APs

In Non Propagated Action potentials there is only slight depolarization of membrane.

In Propagated action potentials following occurs. At resting state the membrane is permeable only to potassium ions. However sodium channels leak. When there is adequate stimulus more number of sodium channels open. Thus Na^+ permeability exceeds potassium ions permeability which leads to adequate depolarization. This results in generation of an action potential.

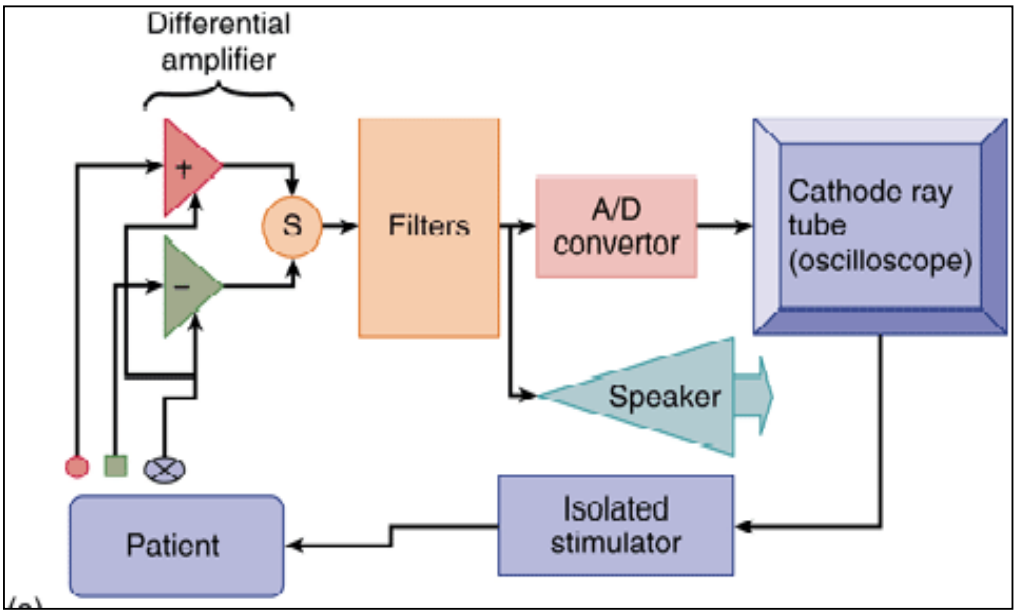
IMPULSE PROPAGATION ACROSS NERVE FIBERS :

In Myelinated nerve fibres the depolarization jumps from one node of Ranvier to the other. This jumping is called 'Saltatory conduction'[52]. It is a rapid process and action potential is conducted upto 50 times faster than unmyelinated fibers.

In unmyelinated fibers, there is continuous conduction which is a slow process.

NERVE CONDUCTION STUDY EQUIPMENT

SCHEMATIC DIAGRAM



NERVE CONDUCTION STUDY

EQUIPMENT:

1. Cathode ray oscilloscope :

A beam of electrons from cathode is focused onto a fluorescent screen as a bright luminous spot. It is made to sweep from left to right in a horizontal plane. The amplified potentials from the tissue under study are applied to the plates above and below the beam to move it in a vertical plane. The movement of the spot traces out the activity as a function of time. The display can be photographed or recorded directly on an ink-writing oscillograph[60].

2. Amplifiers:

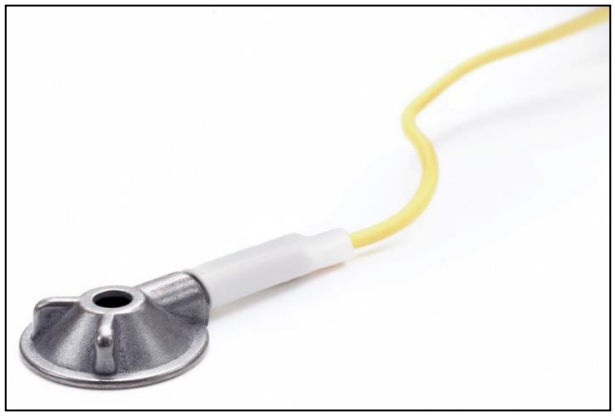
Biological signals are very small because of the intrinsic impedance of the recording electrodes. Electrode-skin contact also reduces the amplitude of potential changes. The amplifier amplifies the signals and minimizes the distortion of waveforms. The sensitivity of the amplifier can be adjusted[60].

3. Filter :

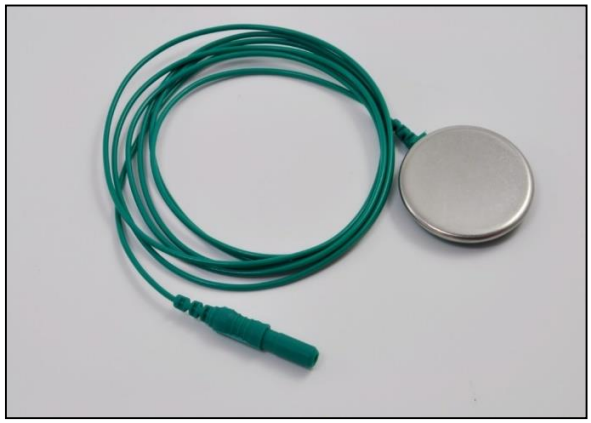
It removes unwanted frequencies from a signal and allows only specific frequencies to pass through[60].

ELECTRODES

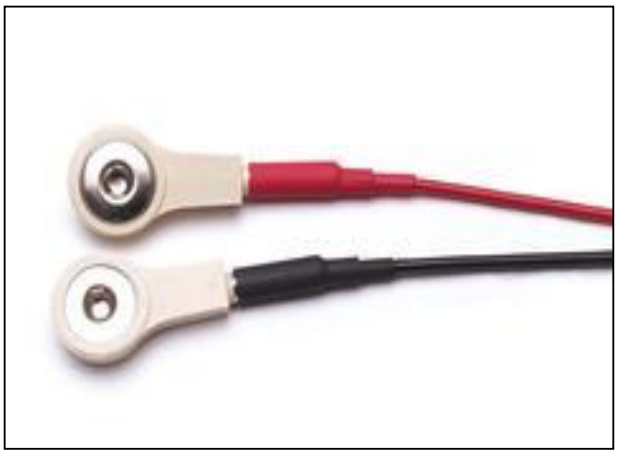
CUP ELECTRODE



GROUND ELECTRODE



DISC ELECTRODE



RING ELECTRODE

4. Averager :

This extracts hidden signals that remains buried in large noise. Eg.Evoked potentials buried in EEG noise, SNAPs buried in EMG noise[60].

5. Stimulators:

Stimulators are of two types:

a) Electrical Stimulators :

They provide constant variable current in single pulse of repeated stimuli.

b) Magnetic Stimulators :

These are used for non-invasive stimulation of motor cortex, spinal cord and peripheral nerves[60].

6. Electrodes:

It is of two types :

a) Recording electrodes

b) Stimulating electrodes

a) Recording electrodes :

Three electrodes are used for recording purposes: active, reference and ground. The action potential is recorded between active and reference electrode. The ground electrode serves as Zero voltage reference point.

They are made of silver, gold, platinum, chromium, Nickel, Stainless steel. When a metal electrode reacts with sweat or electrode paste an

electrochemical reaction occurs which results in electrode polarizing potentials of 100-500 mv[60].

The recording electrodes are of 2 types :

i) Surface electrodes

ii) Concentric needle electrodes.

Surface electrodes are in the forms of discs, cups or rings & record activity from body surface.

Needle electrodes penetrate deep into the particular area of muscle. It is bipolar, one pole formed by shaft and the other by a Teflon coated wire threaded through the shaft[60].

b) Stimulating electrode :

These stimulate the nerves or muscle at a particular point. They are in the form of cups, discs or rings.

FACTORS AFFECTING NERVE CONDUCTION :

1. Physiological
2. Technical

Physiological Factors :

1. Temperature :

Greater the intraneuronal temperature greater is the conduction velocity. It depends on core body temperature. It is found out that 5%

increase in conduction velocity occurs when there is rise in body temperature by a degree within 30^0 - 40^0C range[50].

2. Age :

Age is also an important factor which affects nerve conduction parameters. It is low in infants and children. It attains adult valve by 3 to 5 years of age at which myelination is complete. It remains the same upto 60 years of age after which is declines gradually due to loss of neurons[50].

3. Height :

It has an inverse relationship with nerve conduction velocity. It is because shorter nerves conduct faster than longer nerves of same age group. In tall subject conduction velocity is little slower because of greater axonal tapering and lesser myelination[50].

Limb :

Lower limbs have slower conduction velocity than upper limbs because of

- Greater distal axonal tapering
- Length of nerves
- Lesser myclination
- Shorter internodal distance.
- Lower temperature than upper limbs[50].

TECHNICAL FACTORS :

It can be due to defects in

a) Stimulating System

b) Recording System

STIMULATING SYSTEM[50] :

a) **Faulty location of stimulator :**

Wrong placement of the stimulator over any skin surface leads to decreased stimulation of nerve.

b) **Fat or Edema between Stimulator and Nerve :**

Excessive body fat interferes with proper stimulation of nerve and may lead onto faulty readings.

c) Bridge formation between Cathode & Anode

If there is sweating in the individuals, this leads to shunting of current between cathode and anode. This leads to altered readings.

RECORDING SYSTEMS[50] :

a) Damage in the electrode wire :

The electrode wire is tested for its normal behaviour by asking the subject to contract the muscle to be tested voluntarily with electrode in

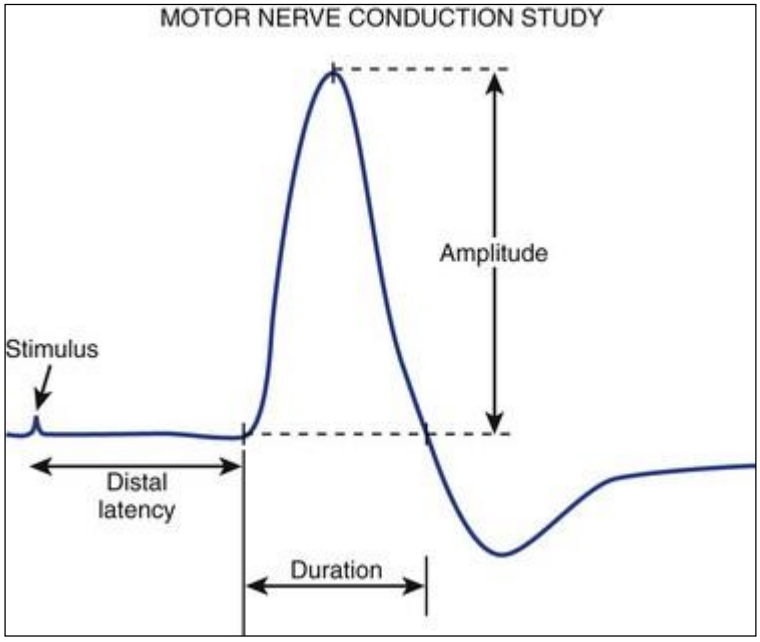
situ. If there is damage in the cable, the stimulus induced muscle twitches causes movement related potentials.

b) Incorrect position of Electrode :

Wrong placement of electrodes over the skin instead of nerve leads to faulty readings.

c) wrongly connected preamplifier

PARAMETERS RECORDED IN A MOTOR NERVE CONDUCTION STUDY :



1. Distal Motor Latency :

It is the time taken for an impulse to travel from distal site of nerve stimulation. It is measured in milliseconds. It is the summation of several events like

a) time taken to depolarize the nerve utilization time.
b) Time taken for the impulse to travel from site of stimulation to motor end plate
c) Residual latency which includes neuromuscular transmission and propagation time along muscle membrane.

2. **Proximal motor latency :**

It is the time taken for an impulse to travel from proximal site of nerve stimulation.

3. **Amplitude :**

It is measured from baseline to the height of positive peak . It corresponds to number of nerve fibers[61].

4. **Duration :**

It is measured from the onset to negative peak or positive peak of the final return of wave to the baseline. It corresponds to density of nerve fibres.

5. **Conduction Velocity :**

It is calculated using the formula

$$NCV = \frac{\text{Distance}}{\text{Proximal latency} - \text{distal latency}}$$

CLINICAL APPLICATIONS OF NCS :

1. NCS is used to locate peripheral nerve disease within single nerves and along the length of nerves.
2. To differentiate nerve lesions from diseases of muscles or NMJ
3. Distinguish axonal degeneration from segmental demyelination[50].

MATERIALS & METHODS

EXAMINATION OF LOWER LIMB NERVE CONDUCTION STUDY

METHODOLOGY

MATERIALS

INCLUSION CRITERIA

- Age :Males 18 – 30 years of age.
- Male footballers who are involved in active play for atleast 3 days a week for the past 5 years are included in the study.
- Control should not be involved in any active sports involving lower limbs and should be of 18 – 30 yrs of age.

EXCLUSION CRITERIA ;

Subjects having diseases affecting nerves of lower limbs such as

Diabetes

Neuromuscular diseases

Leprosy

Drug induced Neuropathy

Malignancy

Myopathies

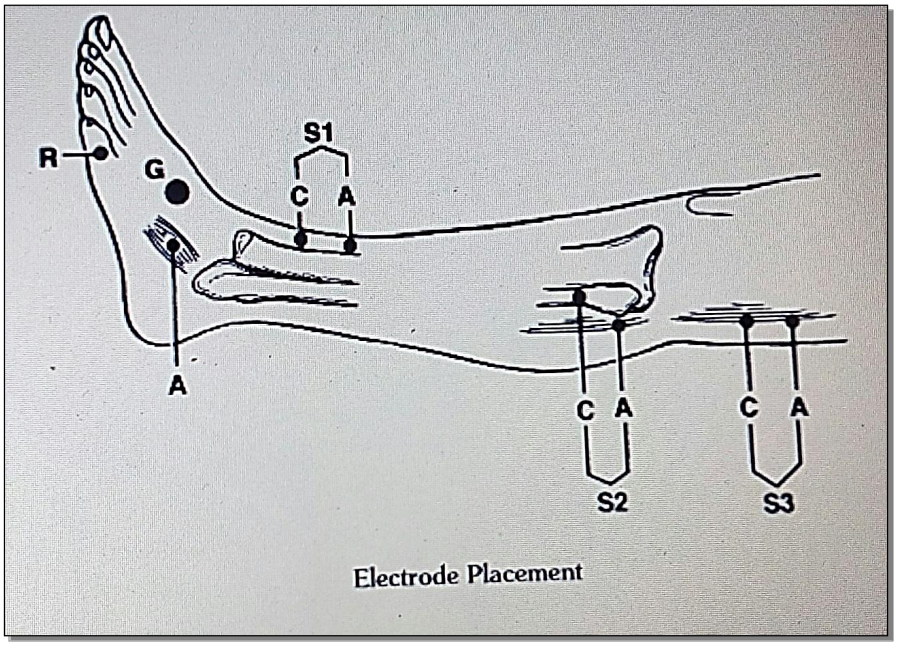
Poliomyelitis

are excluded from the study.

SAMPLE :

A total of 100 subjects 50 football players and 50 controls were included in the study

COMMON PERONEAL NERVE STIMULATION SITES



The picture shows Electrode placement for Common Peroneal Nerve Motor Conduction Study

A - Active Electrode
R – Reference Electrode
G – Ground Electrode
S1 – Distal Stimulation Site
S2 – Proximal Stimulation Sites
C – Cathode
A - Anode

PLACE OF STUDY :

Department of Neurology, Coimbatore medical college hospital.

METHODOLOGY:

50 Football players aged 18-30 years were chosen from the nearby football clubs in and around Coimbatore. 50 controls were normal individuals aged 18-30 years not involved in any active sports of lower limbs.

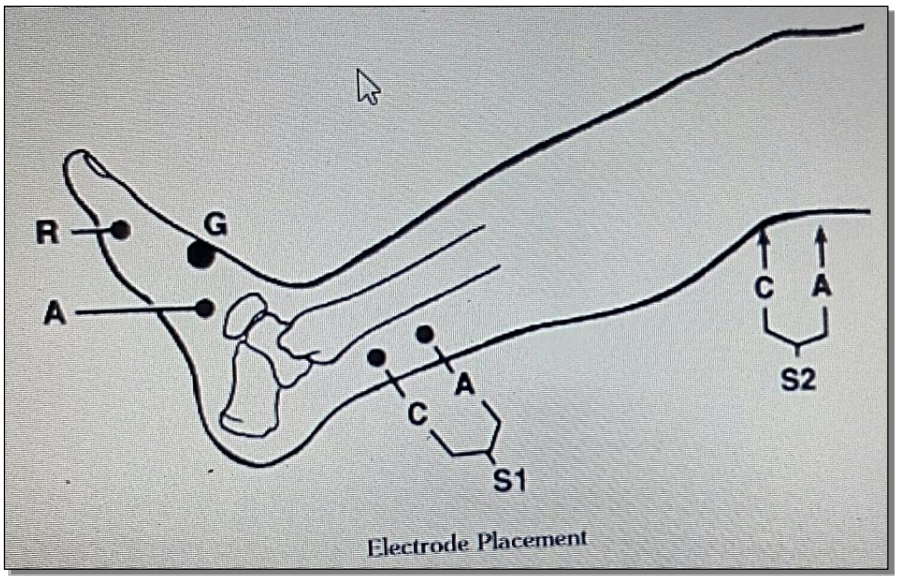
Their height and weight measurements were done. A complete NCS was done in all the subjects using RMS-EMG – EP MARK II using standard protocols and settings. Icon disc surface electrodes were used with surface stimulators. Three types of electrodes were used i.e. active, reference and ground. The ground electrode served as a zero voltage reference point. The nerve conduction velocities to the electrical stimulation of nerves of footballers were compared with normal subjects.

RECORDING OF NCS :

PERONEAL NERVE MOTOR COMPONENT :

The leg – Ankle segment of peroneal nerve is tested. The basic principle is stimulation of common peroneal nerve by a supramaximal stimulus at two points that is neck of fibula and ankle to record a CMAP of extensor digitorum brevis muscle supplied by the nerve using surface electrodes.

TIBIAL NERVE STIMULATION SITES



The picture shows Electrode placement for Tibial Nerve Motor Conduction Study

A - Active Electrode
R – Reference Electrode
G – Ground Electrode
S1 – Distal Stimulation Site
S2 – Proximal Stimulation Sites
C – Cathode
A - Anode

ELECTRODE PLACEMENT :

The deep peroneal motor nerve was examined by stimulating the nerve at two points one at the ankle and the other at the knee. At the ankle the nerve was stimulated lateral to tibialis anterior tendon.

At the knee the nerve was stimulated below the head of fibular bone at the level of its neck. The motor response was recorded from the extensor digitorum brevis with surface electrodes.

TIBIAL NERVE :

The Knee-ankle segment of tibial nerve is tested. Tibial nerve is stimulated using a supra maximal stimulus at two points namely the ankle and popliteal fossa and the CMAP was recorded from abductor hallucis brevis muscle using surface electrodes.

ELECTRODE PLACEMENT :

The tibial motor nerve was examined by stimulating the nerve at two points – ankle and popliteal fossa. At the ankle the electrode was placed posterior to medial malleolus. At the popliteal fossa, the electrode was placed posterior to the knee joint. The nerve was stimulated with bipolar surface electrodes. The recording was carried out over the abductor hallucis brevis muscle with surface electrodes.

The recordings were tabulated with an excel sheet and analysed using SPSS software version 24.

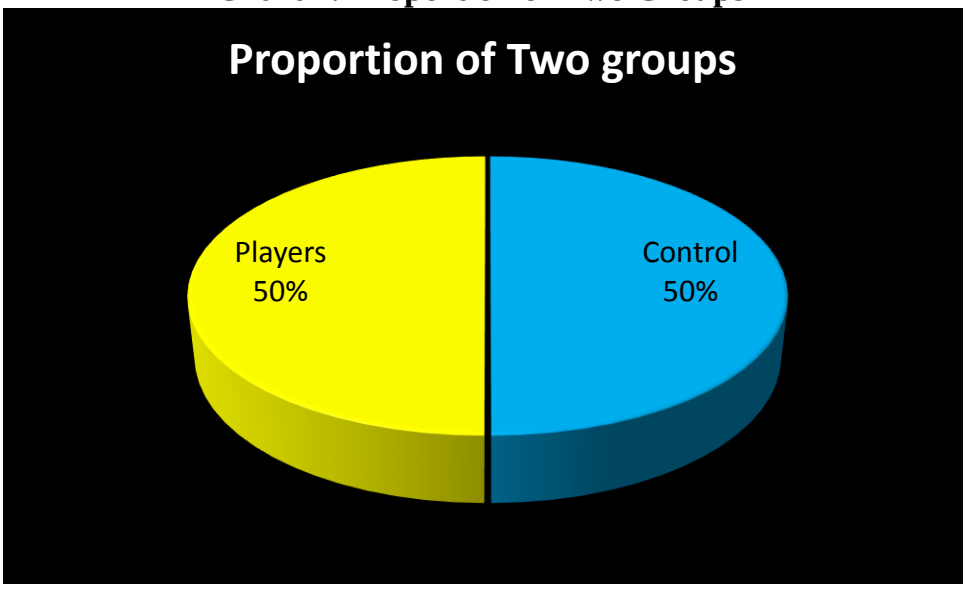
PRECAUTIONS TAKEN :

- Subject is properly instructed and motivated to provide full co-operation.
- Subject is grounded properly.
- Room is made quiet & comfortable
- Subject is made fully relaxed.
- The part to be tested is properly cleaned with spirit to ensure no sweating is there.

RESULTS

RESULTS

Chart 1: Proportion of Two Groups

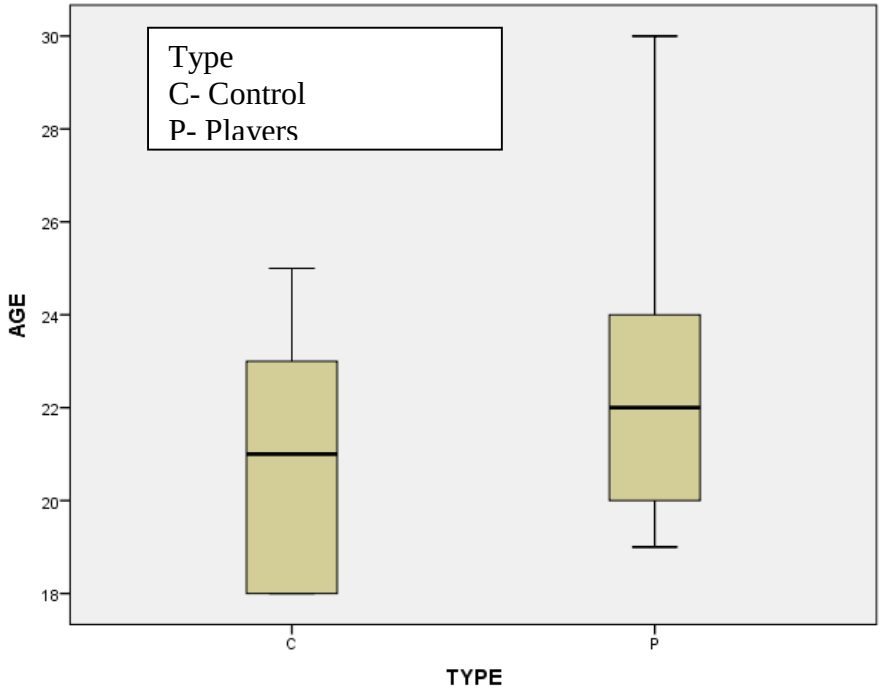


Demographic Profile

Table 1: Age-Wise comparison between two groups

Groups	Numbers	Minimum Age	Maximum Age	Mean	SD
Players	50	19	30	22.46	2.61
Control	50	18	25	20.66	2.26

Chart 2: Age-wise distribution among study population



Anthropometric Profile:

Table 2: Comparison of the two groups based on Anthropometric profile:

Parameters	Players Group (n=50)		Control Group(n=50)		P Value
	Mean	SD	Mean	SD	
Age	22.46	2.61	20.66	2.26	0.06
Height	170.32	3.86	172.96	4.65	0.09
Weight	66.78	3.77	67.74	6.68	0.37
BMI	23.05	1.56	22.69	2.08	0.33

From the above table in the players group the mean age was 22.46 years and standard deviation was 2.61.the mean height was 170.32cm and standard deviation was 3.86.the mean weight was 66.78 years and standard deviation was 3.77.the mean BMI was 23.05 and standard deviation was 1.56.

In the controls group the mean age was 20.66 years and standard deviation was 2.26.the mean height was 172.96cm and standard deviation was 4.65.the mean weight was 67.74kg and standard deviation was 6.68.the mean BMI was 22.69 and standard deviation was 2.08.

On comparing the anthropometric parameters with the nerve conduction parameters, it was found that the p-value for the age, height, weight and BMI were 0.06, 0.09, 0.37 and 0.33 respectively. In all the cases p-value is more than 0.05.This proves that anthropometric measurements does not alter the nerve conduction parameters.

TIBIAL NERVE-ANKLE-FOOT SEGMENT
PROXIMAL LATENCY:
Table 3:DOMINANT LEG:

Time in milliseconds	Players	Control
less than 3	10	16
3 -7.4	32	33
>7.4	8	1

Chart 3: Dominant Leg

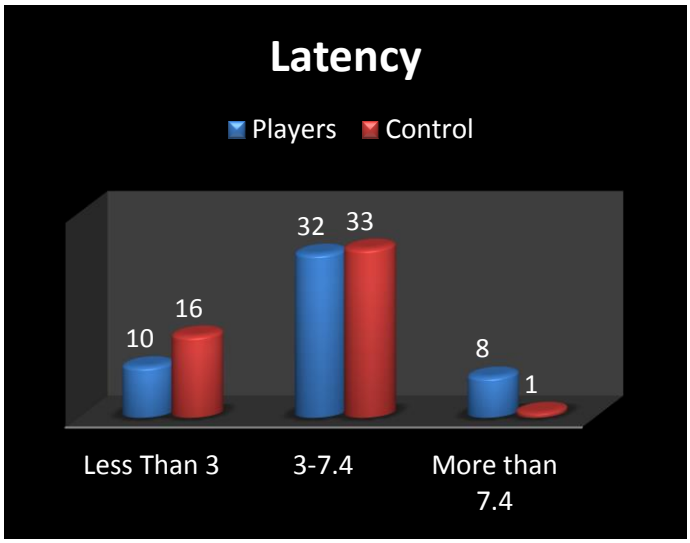
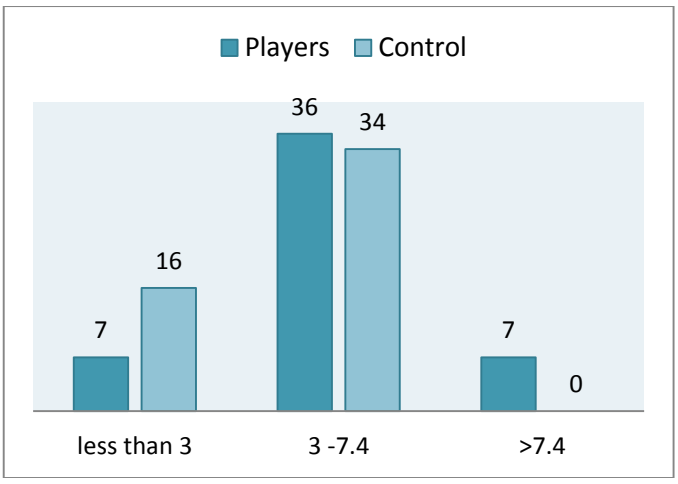


Table 4: NON-DOMINANT LEG:

Time in milliseconds	Players	Control
less than 3	7	16
3 -7.4	36	34
>7.4	7	-

Chart 4: Non-Dominant Leg



TIBIAL NERVE-ANKLE-FOOT SEGMENT
DISTAL LATENCY:
Table 5: DOMINANT LEG:

Time in milliseconds	Players	Control
less than 3	-	-
3 -7.4	40	48
>7.4	10	2

Chart 5: Dominant Leg

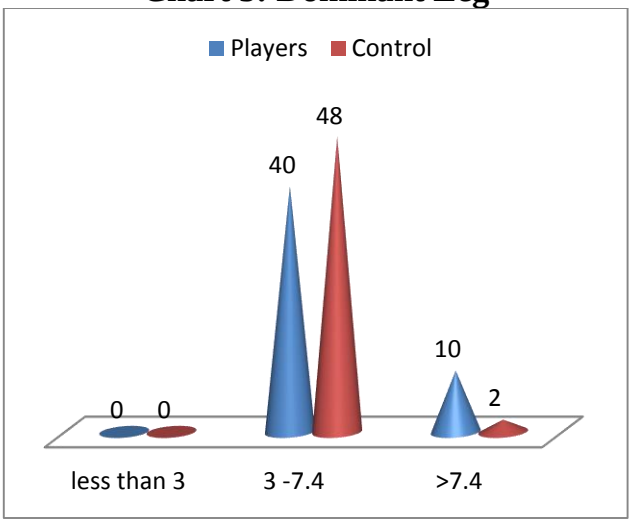
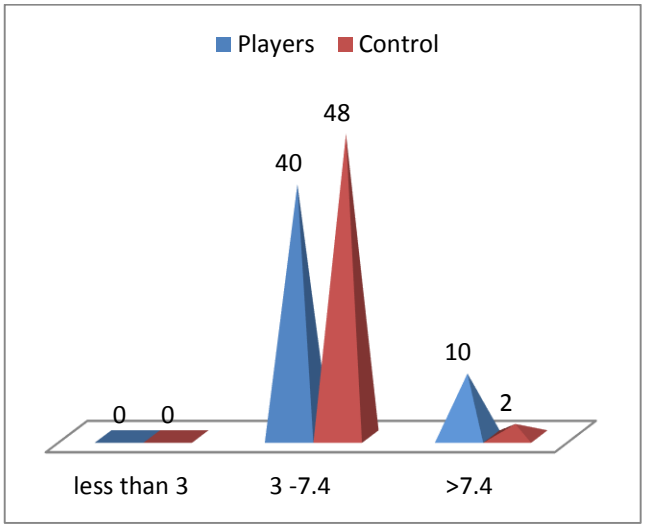


Table 6: NON-DOMINANT LEG:

Time in milliseconds	Players	Control
less than 3	-	-
3 -7.4	40	48
>7.4	10	2

Chart 6: NON-DOMINANT LEG



TIBIAL NERVE-ANKLE-FOOT SEGMENT CONDUCTION VELOCITY:

Table 7: DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	10	2
Normal (38-65)	40	48
More than 65	-	-

Chart 7: DOMINANT LEG

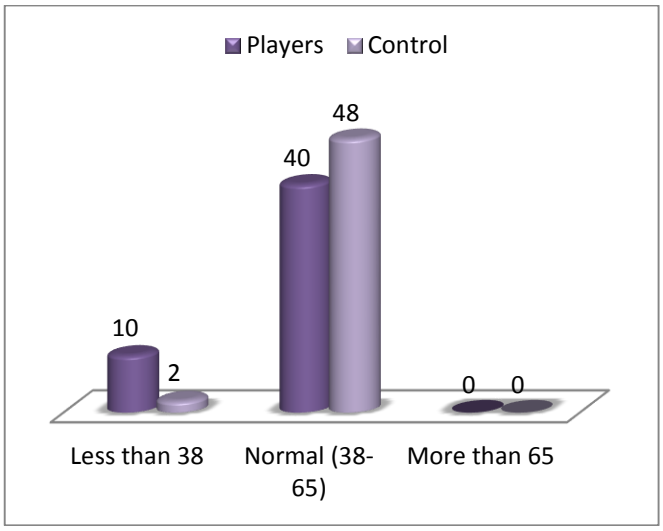
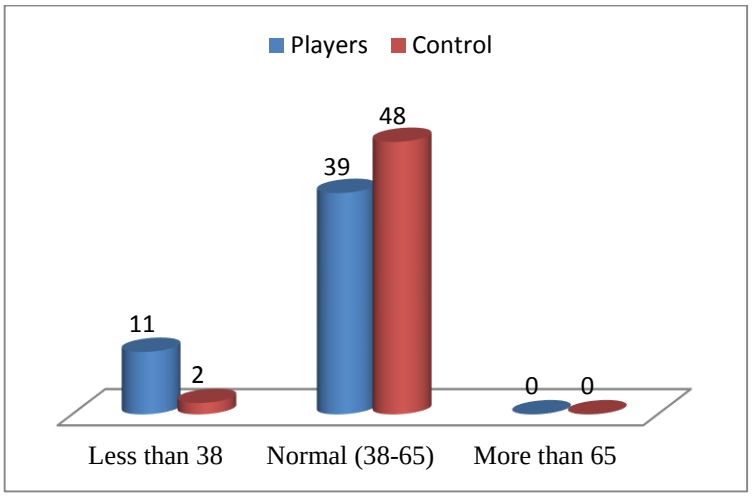


Table 8: NON-DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	11	2
Normal (38-65)	39	48
More than 65	-	-

Chart 8: NON-DOMINANT LEG



TIBIAL NERVE-ANKLE-FOOT SEGMENT

F-WAVE LATENCY:

Table 9: DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	48
>65	10	2

Chart 9: DOMINANT LEG

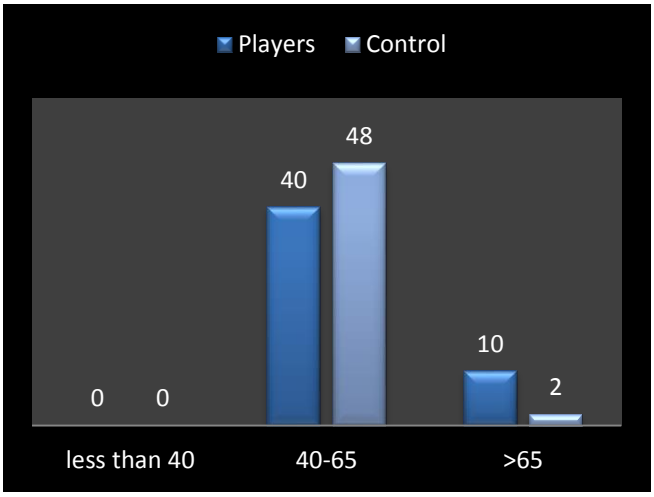
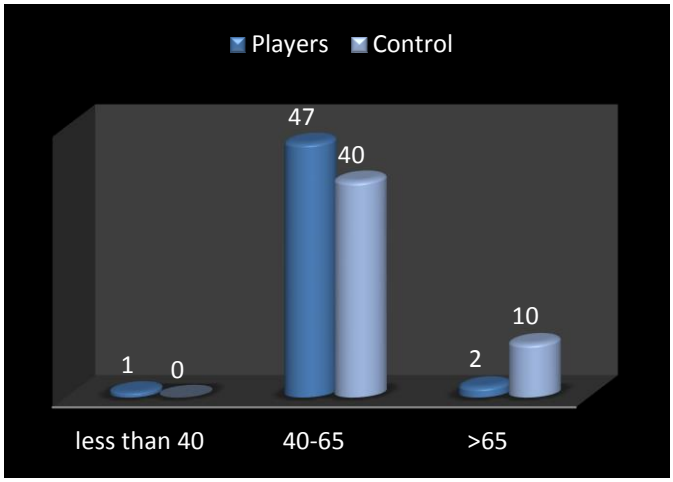


Table 10: NON-DOMINANT LEG

Response in m/s	Players	Control
less than 40	1	0
40-65	47	40
>65	2	10

Chart 10: NON-DOMINANT LEG



TIBIAL NERVE:KNEE TO ANKLE SEGMENT
PROXIMAL LATENCY:
Table 11: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	8	0
3 -7.4	32	48
>7.4	10	2

Chart 11: DOMINANT LEG

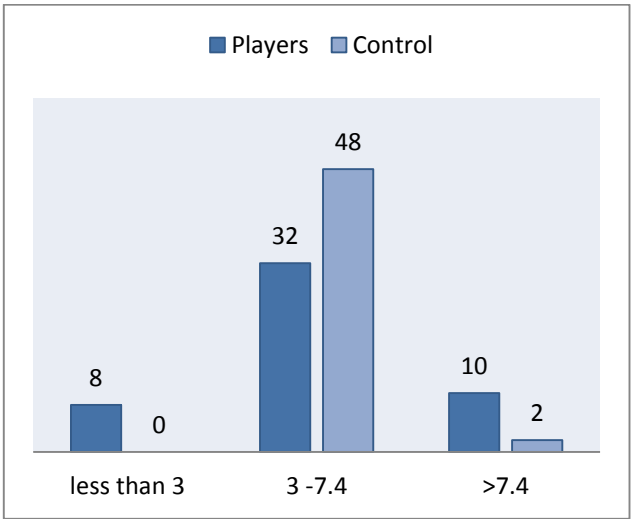
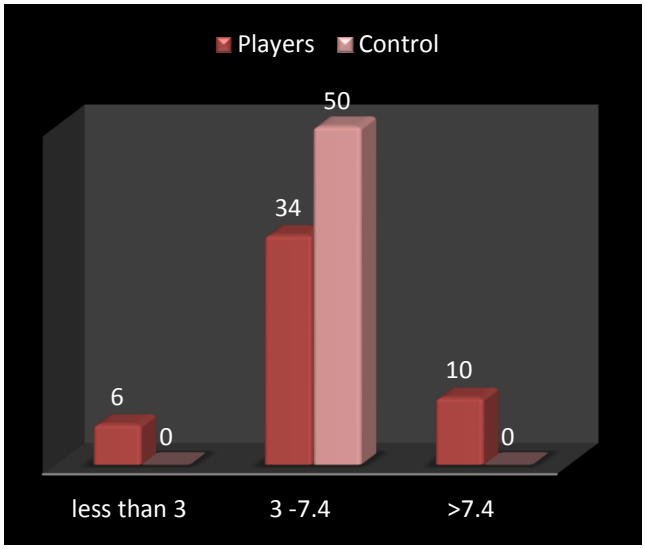


Table 12: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	6	0
3 -7.4	34	50
>7.4	10	0

Table 12: NON-DOMINANT LEG



TIBIAL NERVE:KNEE TO ANKLE SEGMENT

DISTAL LATENCY:

Table 13: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	0	0
3 -7.4	39	47
>7.4	11	3

Chart 13: DOMINANT LEG

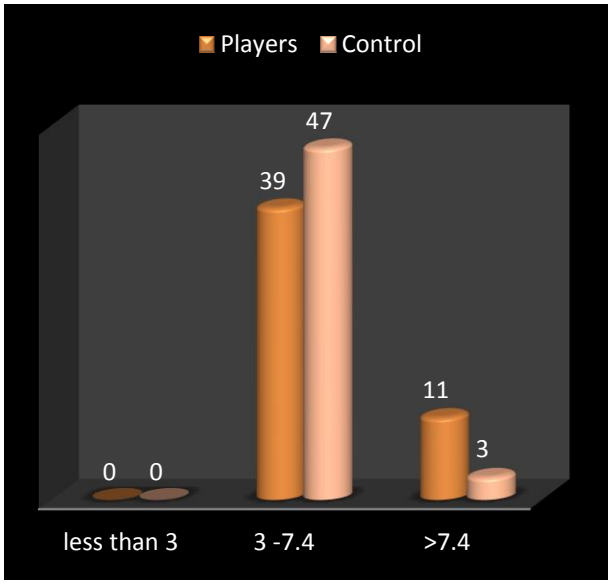
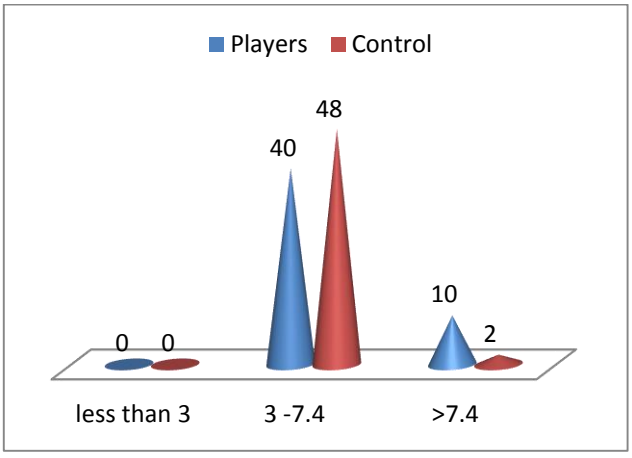


Table 14: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3		
3 -7.4	40	48
>7.4	10	2

Chart 14: NON-DOMINANT LEG



TIBIAL NERVE:KNEE TO ANKLE SEGMENT

CONDUCTION VELOCITY:

Table 15: DOMINANT LEG:

Velocity in m/s	Players	Control
Less than 38	11	2
Normal (38-65)	39	48
Above 65	0	0

Chart 15: DOMINANT LEG:

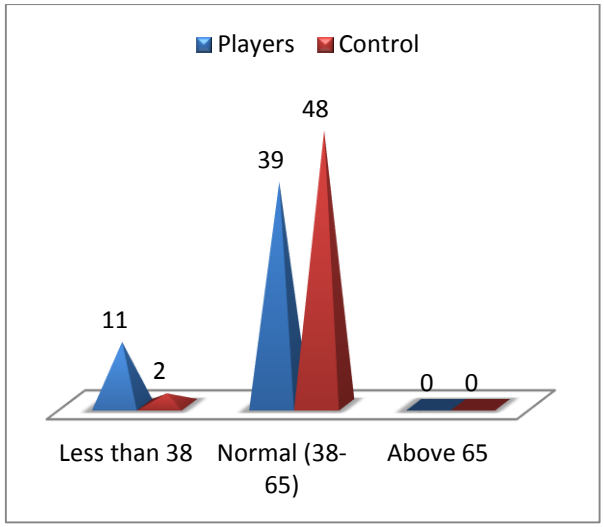
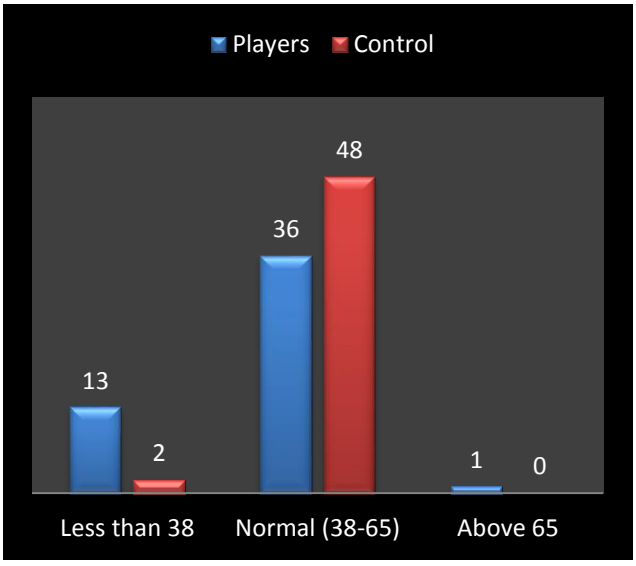


Table 16: NON-DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	13	2
Normal (38-65)	36	48
Above 65	1	0

Chart 16: NON-DOMINANT LEG



TIBIAL NERVE:KNEE TO ANKLE SEGMENT
F-WAVE LATENCY:
Table 17: DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	48
>65	10	2

Chart 17: DOMINANT LEG

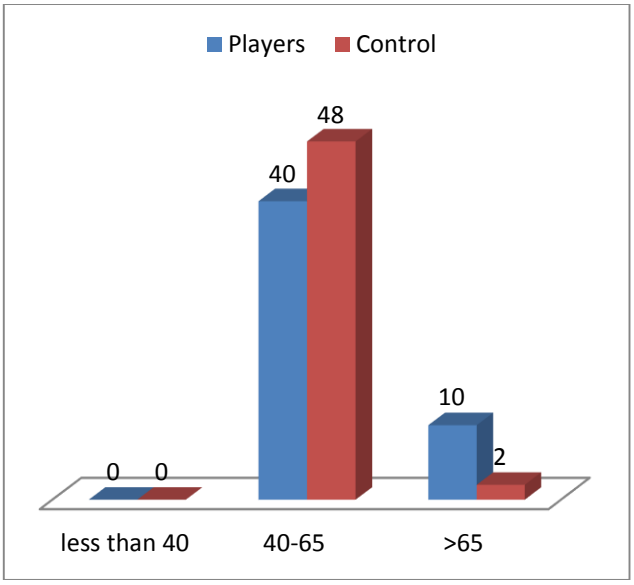
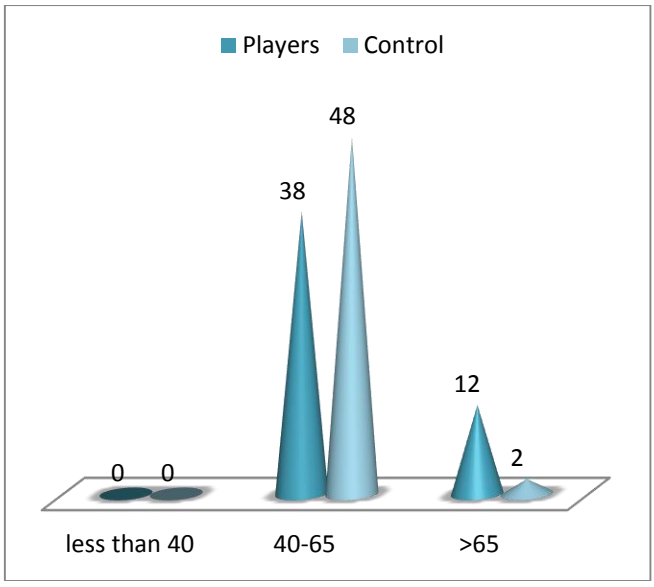


Table 18: NON-DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	38	48
>65	12	2

Chart 18: NON-DOMINANT LEG



COMMON PERONEAL NERVE-ANKLE –FOOT SEGMENT: PROXIMAL LATENCY:

Table 19: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	8	25
3 -7.4	33	24
>7.4	9	1

Chart 19: DOMINANT LEG

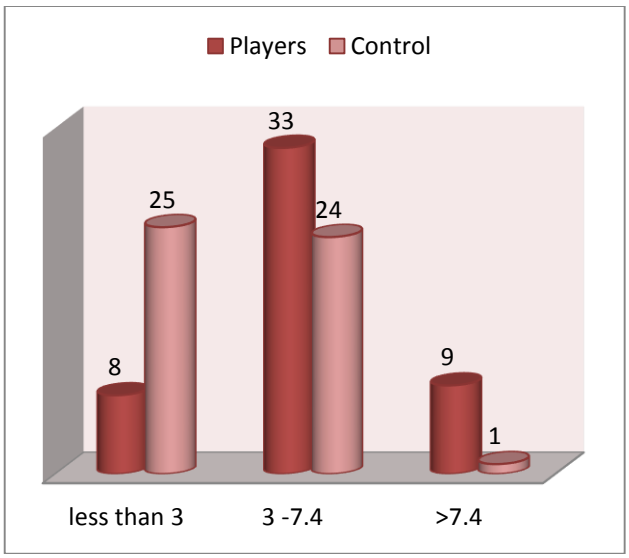
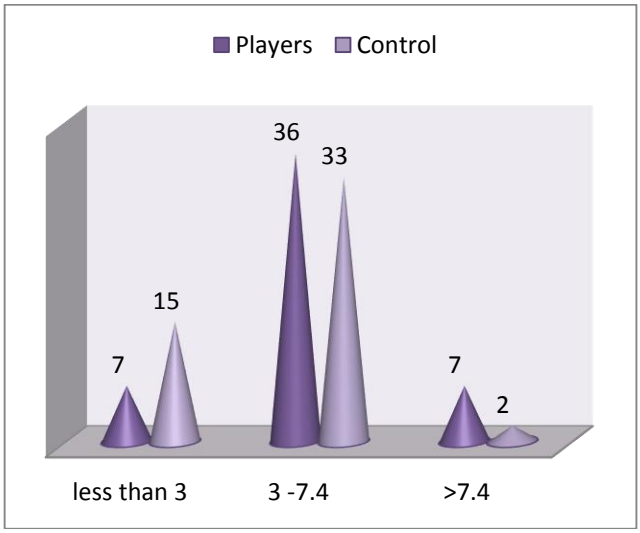


Table 20: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	7	15
3 -7.4	36	33
>7.4	7	2

Chart 20: NON-DOMINANT LEG



COMMON PERONEAL NERVE-ANKLE –FOOT SEGMENT DISTAL LATENCY:

Table 21: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	0	0
3 -7.4	41	48
>7.4	9	2

Chart 21: DOMINANT LEG

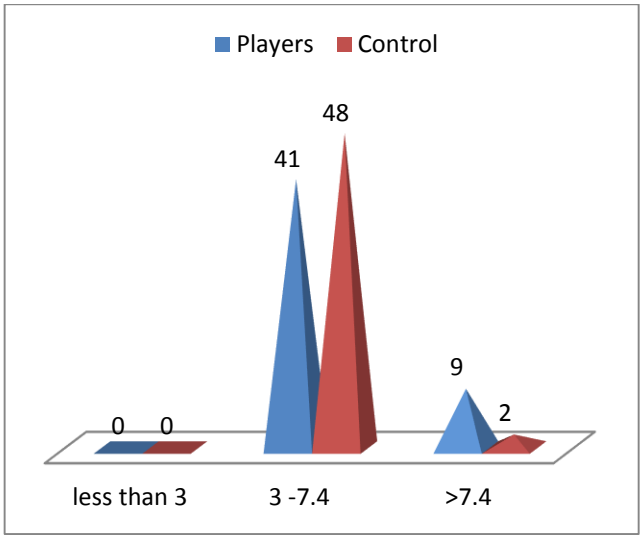
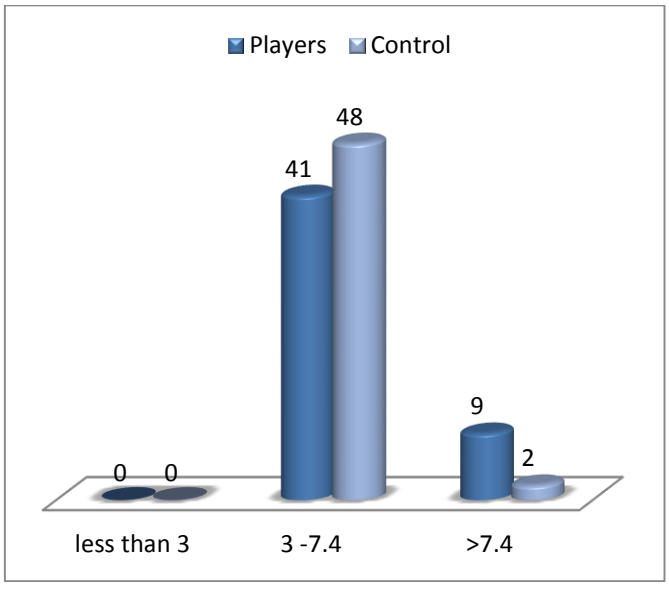


Table 22: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3		
3 -7.4	41	48
>7.4	9	2

Chart 22: NON-DOMINANT LEG



COMMON PERONEAL NERVE-ANKLE –FOOT SEGMENT CONDUCTION VELOCITY:

Table 23: DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	16	2
Normal (38-65)	34	48

Chart 23: DOMINANT LEG

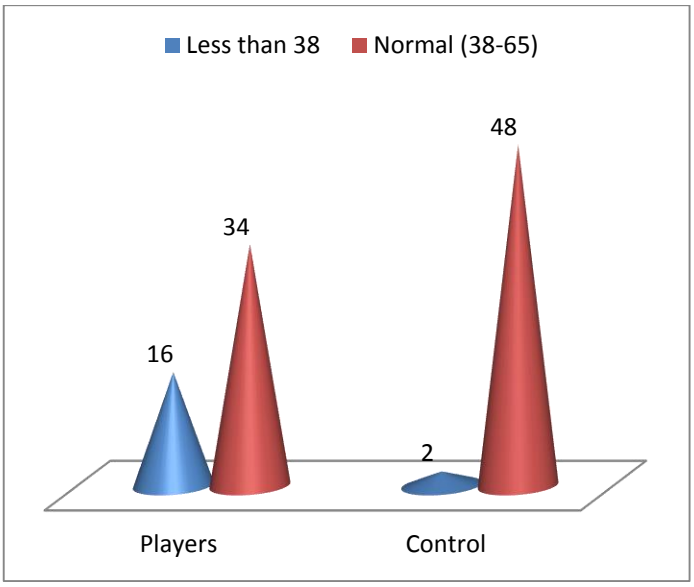
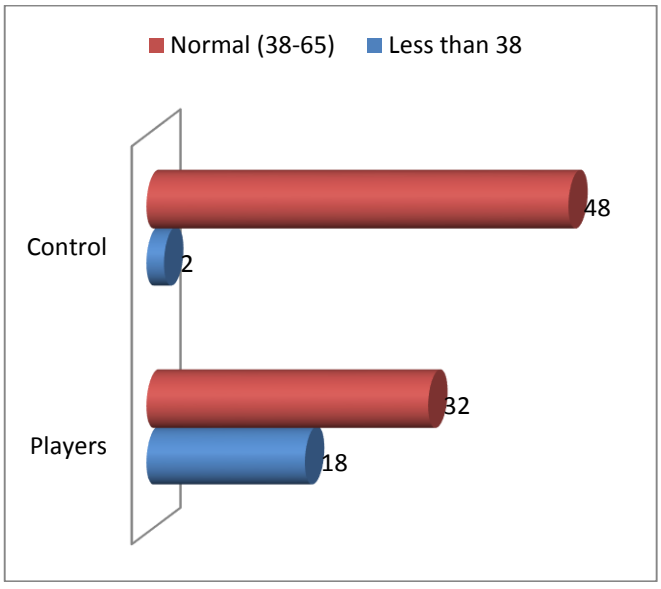


Table 24: NON-DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	18	2
Normal (38-65)	32	48
Above 65	0	0

Chart 24: NON-DOMINANT LEG



COMMON PERONEAL NERVE-ANKLE –FOOT SEGMENT
F-WAVE LATENCY:

Table 25: DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	48
>65	10	2

Chart 25: DOMINANT LEG

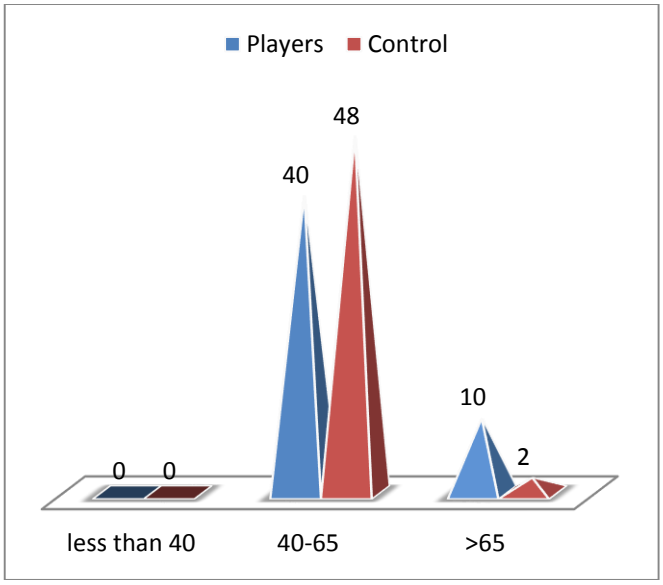
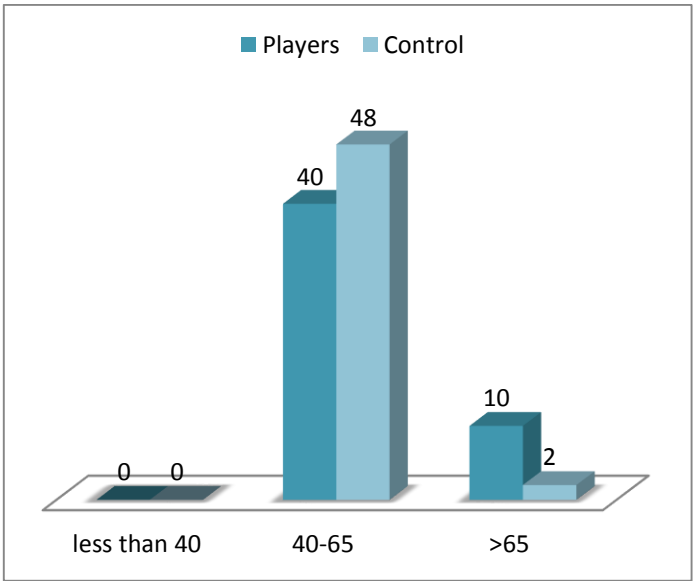


Table 26: NON-DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	48
>65	10	2

Chart 26: NON-DOMINANT LEG



COMMON PERONEAL NERVE:KNEE TO ANKLE SEGMENT:

PROXIMAL LATENCY:

Table 27: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	9	0
3 -7.4	33	48
>7.4	8	2

Chart 27: DOMINANT LEG

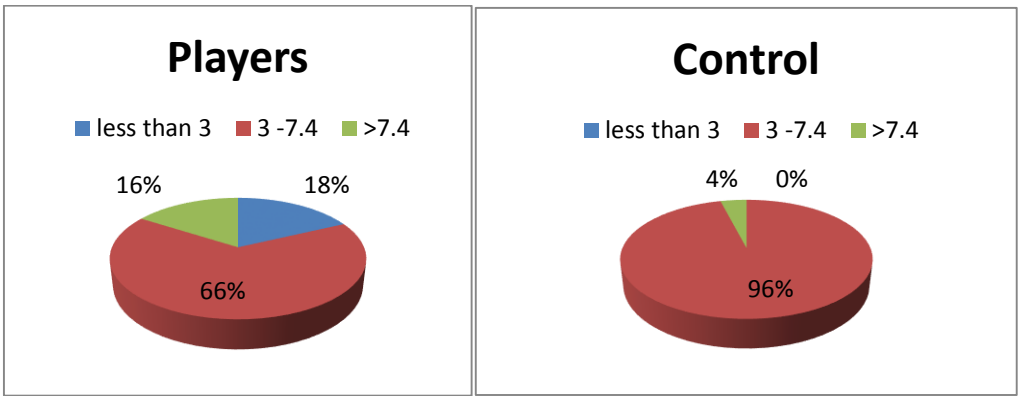
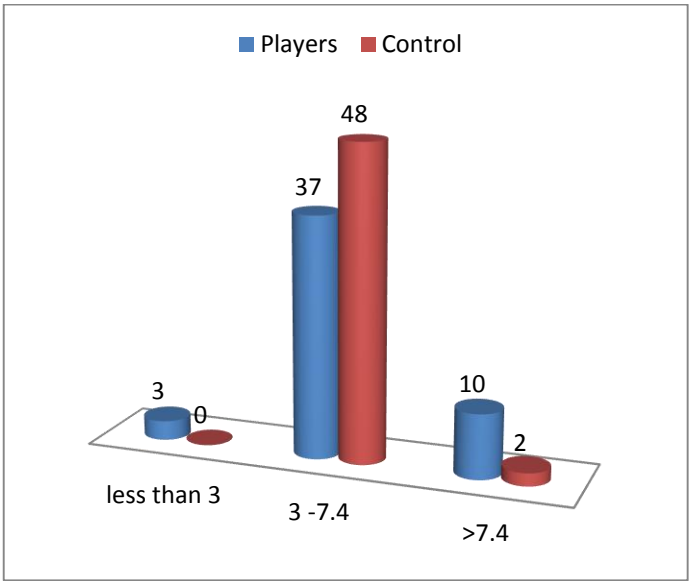


Table 28: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	3	0
3 -7.4	37	48
>7.4	10	2

Chart 28: NON-DOMINANT LEG



COMMON PERONEAL NERVE:KNEE TO ANKLE SEGMENT DISTAL LATENCY:

Table 30: DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	0	0
3 -7.4	40	48
>7.4	10	2

Chart 30: DOMINANT LEG

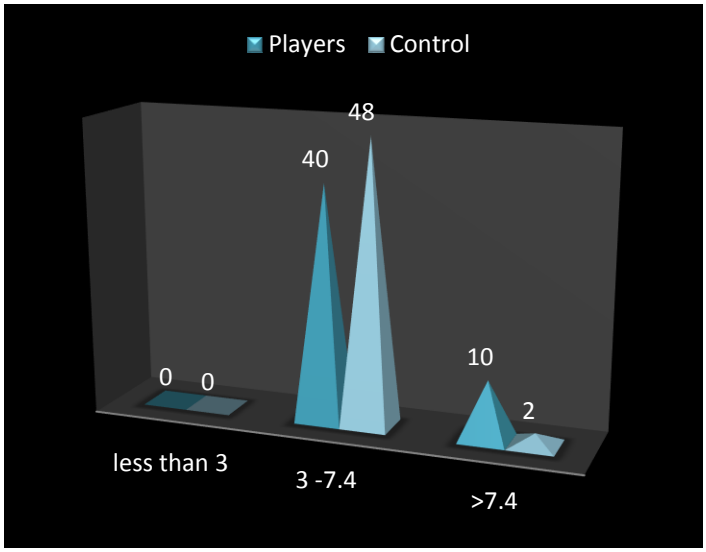
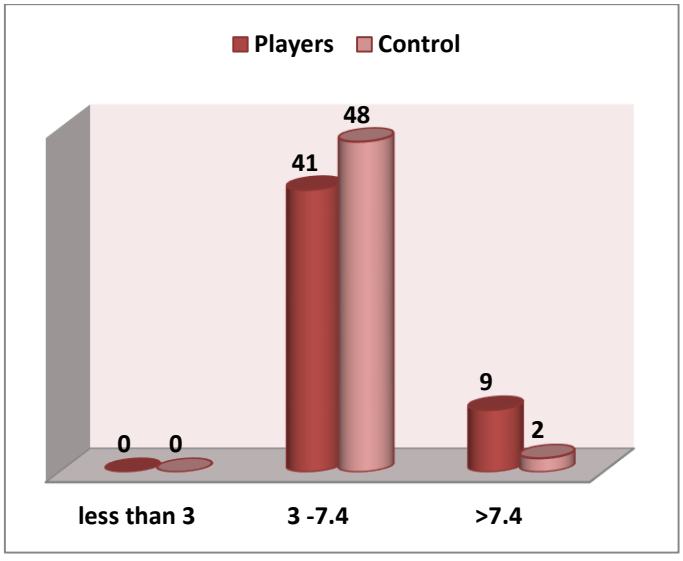


Table 31: NON-DOMINANT LEG

Time in milliseconds	Players	Control
less than 3	0	0
3 -7.4	41	48
>7.4	9	2

Chart 31: NON-DOMINANT LEG



COMMON PERONEAL NERVE:KNEE TO ANKLE SEGMENT CONDUCTION VELOCITY:

Table 32: DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	16	2
Normal (38-65)	31	48
Above 65	3	0

Chart 32: DOMINANT LEG

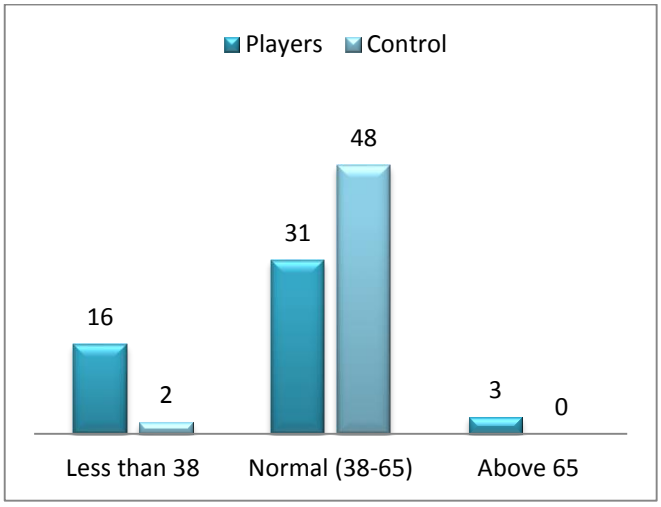
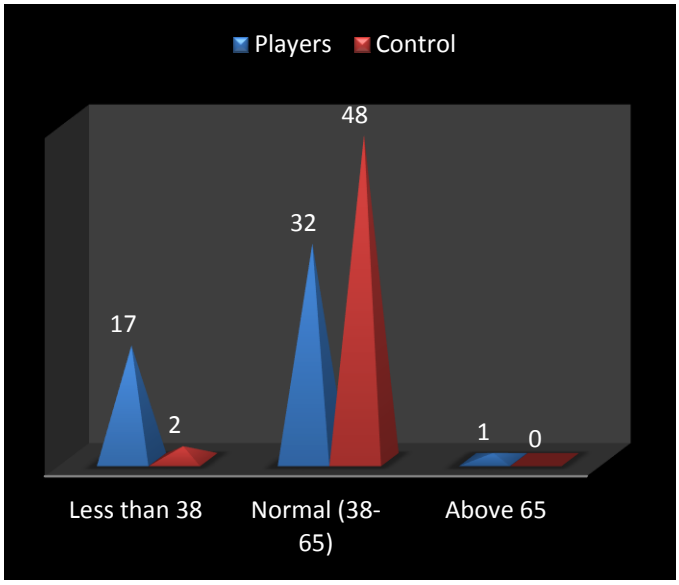


Table 33: NON-DOMINANT LEG

Velocity in m/s	Players	Control
Less than 38	17	2
Normal (38-65)	32	48
Above 65	1	0

Table 33: NON-DOMINANT LEG



COMMON PERONEAL NERVE:KNEE TO ANKLE SEGMENT
F-WAVE LATENCY:

Table 34: DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	46
>65	10	4

Chart 34: DOMINANT LEG

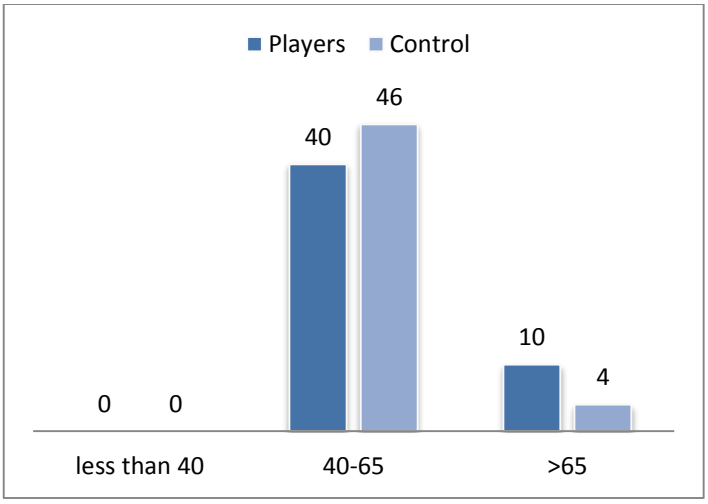
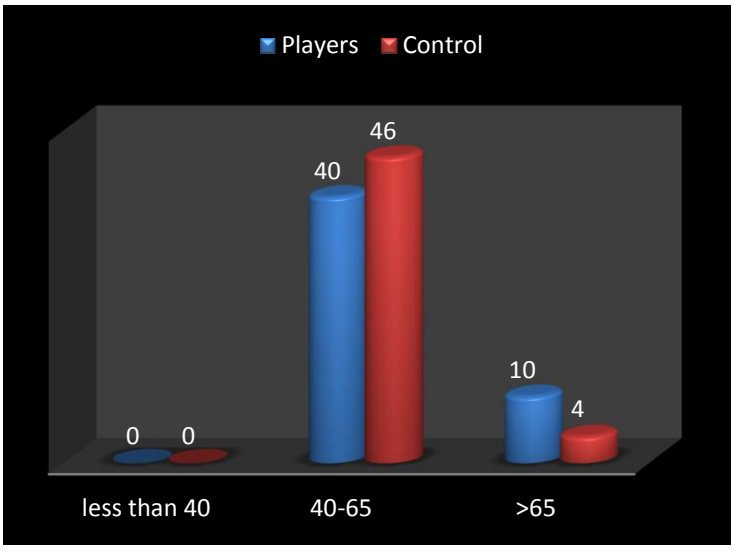


Table 35: NON-DOMINANT LEG

Response in m/s	Players	Control
less than 40	0	0
40-65	40	46
>65	10	4

Chart 35: NON-DOMINANT LEG



CONTROL GROUP:

Table 36: Tibial nerve-ankle to foot segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	3.77	0.83	3.76	0.66
Distal latency	5.68	0.68	5.82	0.63
Amplitude	17.08	4.94	18.38	4.69
distance	100.00	0.00	100.00	0.00
Conduction velocity	51.80	8.01	50.86	9.38
f-wave latency	46.08	11.73	51.04	7.73

For tibial nerve, ankle to foot segment in dominant leg, v Proximal latency was 3.77 and standard deviation was 0.83.Distal latency was 5.68 and standard deviation was 0.68.The amplitude was 17.08 and standard deviation was 4.94.The conduction velocity was 51.80 and standard deviation was 8.01.The f-wave latency was 46.08 and standard deviation was 11.73.

For tibial nerve, ankle to foot segment in non-dominant leg, Proximal latency was 3.76 and standard deviation was 0.66.Distal latency was 5.82 and standard deviation was 0.63.The amplitude was 18.38 and standard deviation was 4.69.The conduction velocity was 50.86 and standard deviation was 9.38.The f-wave latency was 51.04 and standard deviation was 7.73.

CONTROL GROUP

Table 37: Tibial nerve knee to ankle segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	4.80	0.81	4.64	0.63
Distal latency	6.74	0.83	6.82	0.72
amplitude	20.68	3.68	20.76	3.77
distance	360.00	0.00	360.00	0.00
Conduction velocity	53.56	7.42	53.28	8.39
f-wave latency	52.96	7.89	52.82	8.86

For tibial nerve, knee to ankle segment in dominant leg, Proximal latency was 4.80 and standard deviation was 0.81.Distal latency was 6.74 and standard deviation was 0.83.The amplitude was 20.68 and standard deviation was 3.68.The conduction velocity was 53.56 and standard deviation was 7.42.The f-wave latency was 52.96 and standard deviation was 7.89.

For tibial nerve, knee to ankle segment in non-dominant leg, Proximal latency was 4.64 and standard deviation was 0.63.Distal latency was 6.82 and standard deviation was 0.72.The amplitude was 20.76 and standard deviation was 3.77.The conduction velocity was 53.28 and standard deviation was 8.39.The f-wave latency was 52.82 and standard deviation was 8.86.

CONTROL GROUP

Table 38: Common peroneal nerve-ankle to foot segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	3.64	0.94	3.84	0.97
Distal latency	5.66	0.77	5.64	1.12
amplitude	10.04	2.10	9.68	1.82
distance	100.00	0.00	100.00	0.00
Conduction velocity	52.64	8.59	51.44	8.71
f-wave latency	52.98	8.77	51.84	8.62

For common peroneal nerve, ankle to foot segment in dominant leg, Proximal latency was 3.64 and standard deviation was 0.94.Distal latency was 5.66 and standard deviation was 0.77.The amplitude was 10.04 and standard deviation was 2.10.The conduction velocity was 52.64 and standard deviation was 8.59.The f-wave latency was 52.98 and standard deviation was 8.77.

For common peroneal nerve, ankle to foot segment in non-dominant leg, Proximal latency was 3.84 and standard deviation was 0.97.Distal latency was 5.64 and standard deviation was 1.12.The amplitude was 9.68 and standard deviation was 1.82.The conduction velocity was 51.44 and standard deviation was 8.71.The f-wave latency was 51.84 and standard deviation was 8.62.

CONTROL GROUP

Table 39: Common peroneal nerve-knee to ankle segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	4.66	0.94	4.62	0.85
Distal latency	6.56	0.70	6.58	0.73
amplitude	9.38	1.80	9.56	2.13
distance	380.00	0.00	380.00	0.00
Conduction velocity	52.48	8.69	51.58	8.68
F-wave latency	51.66	8.39	51.12	8.56

For common peroneal nerve, knee to ankle segment in dominant leg, Proximal latency was 4.66 and standard deviation was 0.94.Distal latency was 6.56 and standard deviation was 0.70.The amplitude was 9.38 and standard deviation was 1.80.The conduction velocity was 52.48 and standard deviation was 8.69.The f-wave latency was 51.66 and standard deviation was 8.39.

For common peroneal nerve, knee to ankle segment in non-dominant leg, Proximal latency was 4.62 and standard deviation was 0.85.Distal latency was 6.58 and standard deviation was 0.73.The amplitude was 9.56 and standard deviation was 2.13.The conduction velocity was 51.58 and standard deviation was 8.68.The f-wave latency was 51.12 and standard deviation was 8.56.

PLAYERS GROUP

Table 40: Tibial nerve: Ankle to foot segment:

NCS parameters	Dominant		Non-Dominant	
	Mean	**SD**	**Mean**	**SD**
Proximal latency	4.80	1.80	4.76	1.74
Distal latency	6.92	1.65	7.02	1.42
amplitude	15.38	5.76	15.58	5.33
distance	100.00	0.00	100.00	0.00
Conduction velocity	44.40	11.33	45.32	11.35
F-wave latency	46.00	11.73	46.42	11.40

For tibial nerve, ankle to foot segment in dominant leg, Proximal latency was 4.80 and standard deviation was 1.80.Distal latency was 6.92 and standard deviation was 1.65.The amplitude was 15.38 and standard deviation was 5.76.The conduction velocity was 44.40 and standard deviation was 11.33.The f-wave latency was 46.00 and standard deviation was 11.73.

For tibial nerve, ankle to foot segment in non-dominant leg, Proximal latency was 4.76 and standard deviation was 1.74.Distal latency was 7.02 and standard deviation was 1.42.The amplitude was 15.58 and standard deviation was 5.33.The conduction velocity was 45.32 and standard deviation was 11.35.The f-wave latency was 46.42 and standard deviation was 11.40.

PLAYERS GROUP

Table 41: Tibial nerve: Knee to ankle segment:

NCS parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	4.88	1.99	5.08	2.08
Distal latency	6.98	1.86	7.14	2.07
amplitude	15.50	5.25	16.28	5.28
distance	360.00	0.00	360.00	0.00
Conduction velocity	44.85	11.52	45.62	11.39
f-wave latency	46.58	11.06	47.02	11.77

For tibial nerve, knee to ankle segment in dominant leg, Proximal latency was 4.88 and standard deviation was 1.99.Distal latency was 6.98 and standard deviation was 1.86.The amplitude was 15.50 and standard deviation was 5.25.The conduction velocity was 44.85 and standard deviation was 11.52.The f-wave latency was 46.58 and standard deviation was 11.06.

For tibial nerve, knee to ankle segment in non- dominant leg, Proximal latency was 5.08 and standard deviation was 2.08.Distal latency was 7.14 and standard deviation was 2.07.The amplitude was 16.28 and standard deviation was 5.28.The conduction velocity was 45.62 and standard deviation was 11.39.The f-wave latency was 47.02 and standard deviation was 11.77.

PLAYERS GROUP

Table 42: Common peroneal nerve: ankle to foot segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	5.12	2.31	4.92	2.21
Distal latency	6.80	2.19	6.52	1.48
amplitude	7.24	2.88	7.62	2.78
Distance	100.00	0.00	100.00	0.00
Conduction velocity	43.58	13.71	43.52	13.05
F wave latency	44.39	11.58	45.10	11.99

For common peroneal nerve, ankle to foot segment in dominant leg, Proximal latency was 5.12 and standard deviation was 2.31.Distal latency was 6.80 and standard deviation was 2.19.The amplitude was 7.24 and standard deviation was 2.88.The conduction velocity was 43.58 and standard deviation was 13.71.The f-wave latency was 44.39 and standard deviation was 11.58.

For common peroneal nerve, ankle to foot segment in non-dominant leg, Proximal latency was 4.92 and standard deviation was 2.21.Distal latency was 6.52 and standard deviation was 1.48.The amplitude was 7.62 and standard deviation was 2.78.The conduction velocity was 43.52 and standard deviation was 13.05.The f-wave latency was 45.10 and standard deviation was 11.99.

PLAYERS GROUP

Table 43: Common peroneal nerve: knee to ankle segment:

NCS Parameters	Dominant		Non-Dominant	
	Mean	SD	Mean	SD
Proximal latency	5.12	2.48	5.10	2.13
Distal latency	6.72	2.05	6.34	1.37
amplitude	7.32	2.51	7.44	2.72
distance	380.00	0.00	380.00	0.00
Conduction velocity	44.06	13.89	44.26	13.74
f-wave latency	44.64	11.58	45.82	12.67

For common peroneal nerve, knee to ankle segment in dominant leg, Proximal latency was 5.12 and standard deviation was 2.48.Distal latency was 6.72 and standard deviation was 2.05.The amplitude was 7.32 and standard deviation was 2.51.The conduction velocity was 44.06 and standard deviation was 13.89.The f-wave latency was 44.64 and standard deviation was 11.58.

For common peroneal nerve, knee to ankle segment in non-dominant leg, Proximal latency was 5.10 and standard deviation was 2.13.Distal latency was 6.34 and standard deviation was 1.37.The amplitude was 7.44 and standard deviation was 2.72.The conduction velocity was 44.26 and standard deviation was 13.74.The f-wave latency was 45.82 and standard deviation was 12.67.

Comparison between the two groups:

Table 44: TIBIAL NERVE:ANKLE TO FOOT SEGMENT:

Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	4.80	1.80	3.76	0.83	**<0.05**
Distal latency	6.92	1.65	5.68	0.68	**<0.05**
amplitude	15.38	5.76	17.08	4.94	0.12
distance	100.00	0.00	100.00	0.00	-
Conduction velocity	44.40	11.33	51.80	8.01	**<0.05**
f-wave latency	46.00	11.73	51.08	7.78	**<0.05**

On comparison, there was a significant delay in the latencies and conduction velocities of tibial nerve(ankle to foot segment) of dominant limb of players when compared to controls. p-value<0.05

Table 45: Non-Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	4.76	1.74	3.76	0.65	**<0.05**
Distal latency	7.02	1.42	5.82	0.62	**<0.05**
amplitude	15.58	5.33	18.38	4.68	0.06
distance	100.00	0.00	100.00	0.00	-
Conduction velocity	45.32	11.35	50.86	9.38	**<0.05**
f-wave latency	46.42	11.40	51.50	9.25	**<0.05**

On comparison, there was a significant delay in the latencies and conduction velocities of tibial nerve(ankle to foot segment) of non-dominant limb of players when compared to controls. p-value<0.05

TIBIAL NERVE-KNEE TO ANKLE SEGMENT:

Table 46: Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	4.88	1.99	4.80	0.80	0.79
Distal latency	6.98	1.86	6.74	1.60	0.33
amplitude	15.50	5.25	20.68	3.68	**<0.05**
distance	360.00	0.00	360.00	0.00	-
Conduction velocity	44.85	11.52	53.56	7.42	**<0.05**
f-wave latency	46.58	11.06	52.96	7.89	**<0.05**

On comparison, there was a significant delay in the f-wave latencies and conduction velocities of tibial nerve(knee to ankle segment) of dominant limb of players when compared to controls. p-value<0.05

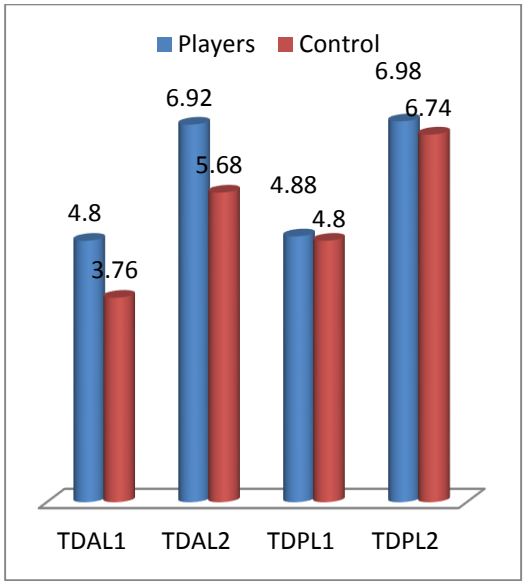
Table 47: Non-Dominant Limb

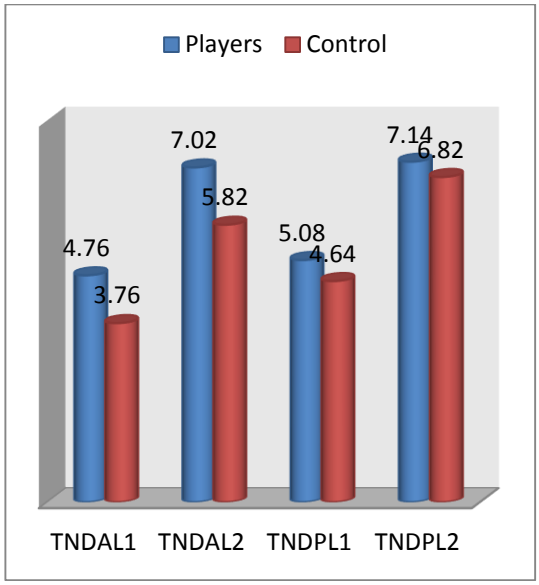
NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	5.08	2.08	4.64	0.63	0.16
Distal latency	7.14	2.07	6.82	0.72	0.30
amplitude	16.28	5.28	20.76	3.77	**<0.05**
distance	360.00	0.00	360.00	0.00	-
Conduction velocity	45.62	11.39	53.28	8.39	**<0.05**
f-wave latency	47.02	11.77	52.82	8.85	**<0.05**

On comparison, there was a significant delay in the f-wave latencies and conduction velocities of tibial nerve(knee to ankle segment) of non-dominant limb of players when compared to controls. p-value<0.05

Chart 36 & 37:

Dominant Limb – Tibial N Latency Non-dominant limb-Tibial N Latency



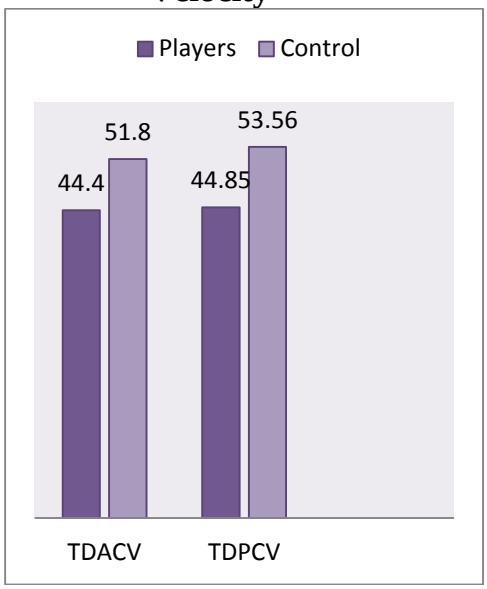


In both dominant limb and non-dominant limb ,the mean value of proximal latency of tibial nerve of players was higher than that of controls. This shows a slower nerve conduction in players than controls.

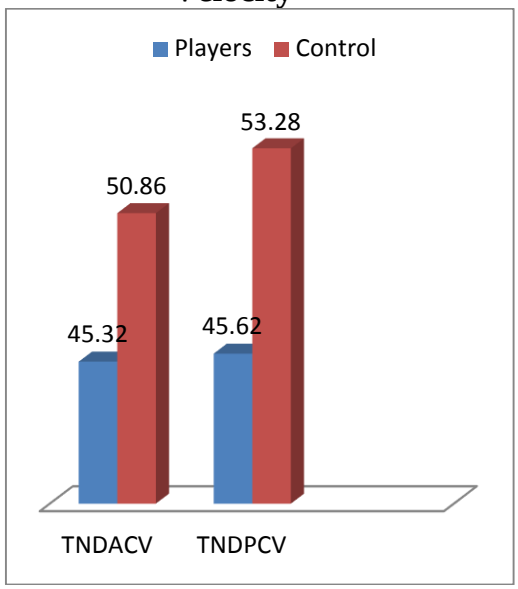
In both dominant limb and non-dominant limb, the mean value of distal latency of tibial nerve of players was higher than that of controls. This shows a slower nerve conduction in players than controls.

Chart 38 & 39:

Dominant limb – Conduction Velocity

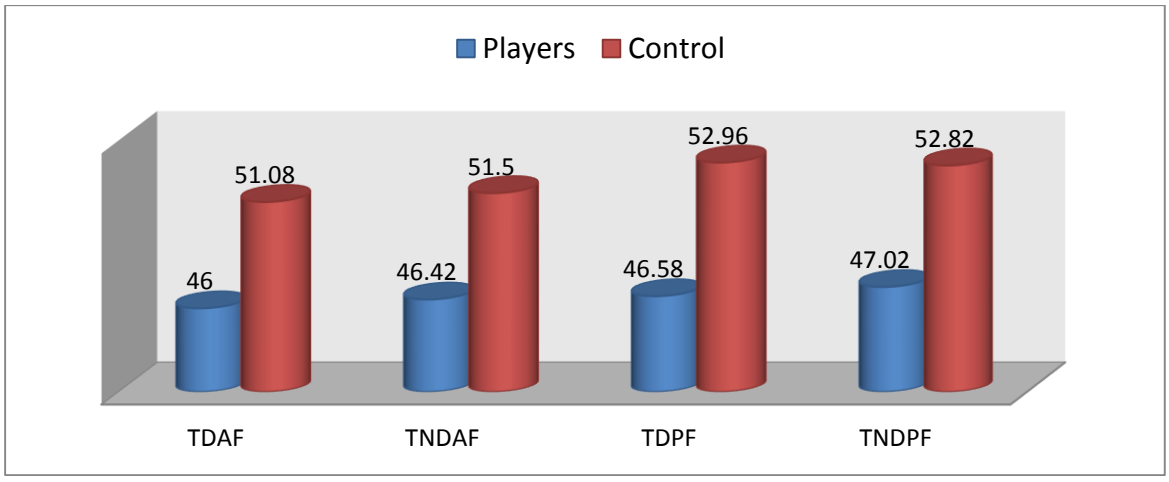


Non-dominant limb-conduction Velocity



In both dominant limb and non-dominant limb, the mean value of conduction velocity of tibial nerve of players was lower than that of controls. This shows a decreased nerve conduction in players than controls

Chart 40: F-WAVE LATENCY



In both dominant limb and non-dominant limb, the mean value of f-wave latency of tibial nerve of players was lower than that of controls. This shows a decreased nerve conduction in players than controls.

COMMON PERONEAL NERVE:ANKLE TO FOOT SEGMENT:

Table 48: Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	5.12	2.31	3.64	0.94	**<0.05**
Distal latency	6.80	2.19	5.66	0.77	**<0.05**
amplitude	7.24	2.88	10.04	2.10	**<0.05**
distance	100.00	0.00	100.00	0.00	-
Conduction velopcity	43.58	13.71	52.64	8.59	**<0.05**
F-wave latency	44.39	11.58	52.98	8.77	**<0.05**

On comparison, there was a significant delay in the latencies and conduction velocities of common peroneal nerve(ankle to foot segment) of dominant limb of players when compared to controls. p-value<0.05

Table 49: Non-Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	4.92	2.21	3.84	0.97	**<0.05**
Distal latency	6.52	1.48	5.64	1.12	**<0.05**
amplitude	7.62	2.78	9.68	1.82	**<0.05**
distance	100.00	0.00	100.00	0.00	-
Conduction velocity	43.52	13.05	51.44	8.71	**<0.05**
f-wave latency	45.10	11.99	51.84	8.62	**<0.05**

On comparison, there was a significant delay in the latencies and conduction velocities of common peroneal nerve(ankle to foot segment) of non-dominant limb of players when compared to controls. p-value<0.05

COMMON PERONEAL NERVE-KNEE TO ANKLE SEGMENT:

Table 50: Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	5.12	2.48	4.66	0.93	0.23
Distal latency	6.72	2.05	6.56	0.70	0.61
amplitude	7.32	2.51	9.38	1.86	**<0.05**
distance	380.00	0.00	380.00	0.00	-
Conduction velocity	44.06	13.89	52.48	8.69	**<0.05**
f-wave latency	44.64	11.58	51.66	8.39	**<0.05**

On comparison, there was a significant delay in the f-wave latencies and conduction velocities of common peroneal nerve(knee to ankle segment) of dominant limb of players when compared to controls. p-value<0.05

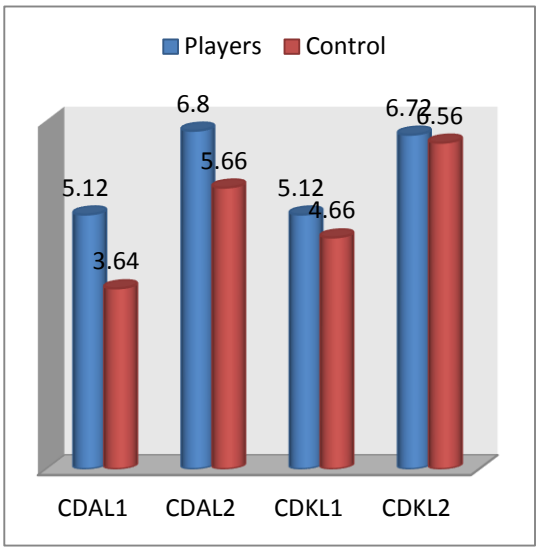
Table 51: Non-Dominant Limb

NCS Parameters	Players		Control		P value
	Mean	SD	Mean	SD	
Proximal latency	5.10	2.13	4.62	0.85	0.14
Distal latency	6.34	1.37	6.58	0.73	0.28
amplitude	7.44	2.72	9.56	2.13	**<0.05**
Distance	380.00	0.00	380.00	0.00	-
Conduction velocity	44.26	13.74	51.58	8.68	**<0.05**
f-wave latency	45.82	12.67	51.12	8.55	**<0.05**

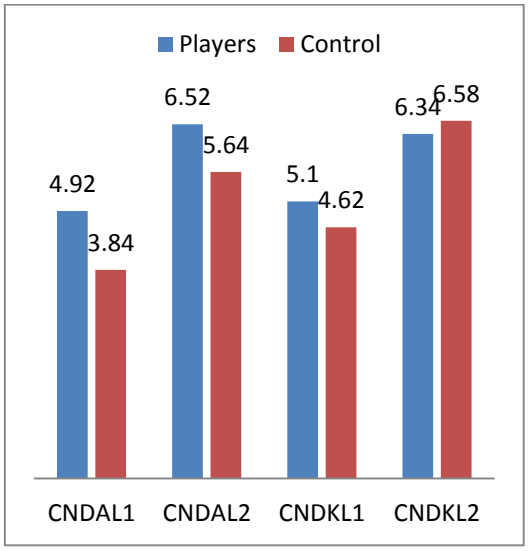
On comparison, there was a significant delay in the f-wave latencies and conduction velocities of common peroneal nerve(knee to ankle segment) of non- dominant limb of players when compared to controls. p-value<0.05

Chart 41 & 42:

Dominant Limb-Latency



Non-Dominant Limb –Latency

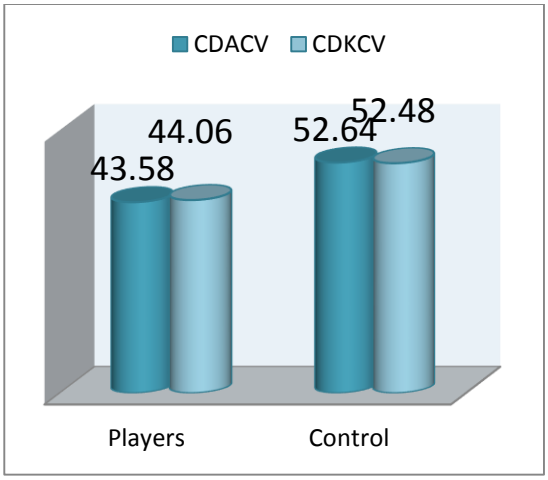


In both dominant limb and non-dominant limb, the mean value of proximal latency of common peroneal nerve of players was higher than that of controls. This shows a slower nerve conduction in players than controls.

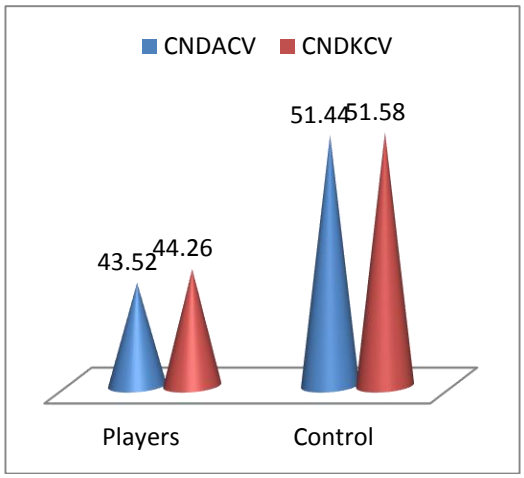
In both dominant limb and non-dominant limb, the mean value of distal latency of common peroneal nerve of players was higher than that of controls. This shows a slower nerve conduction in players than controls.

Chart 43 & 44: CONDUCTION VELOCITY

Dominant Limb

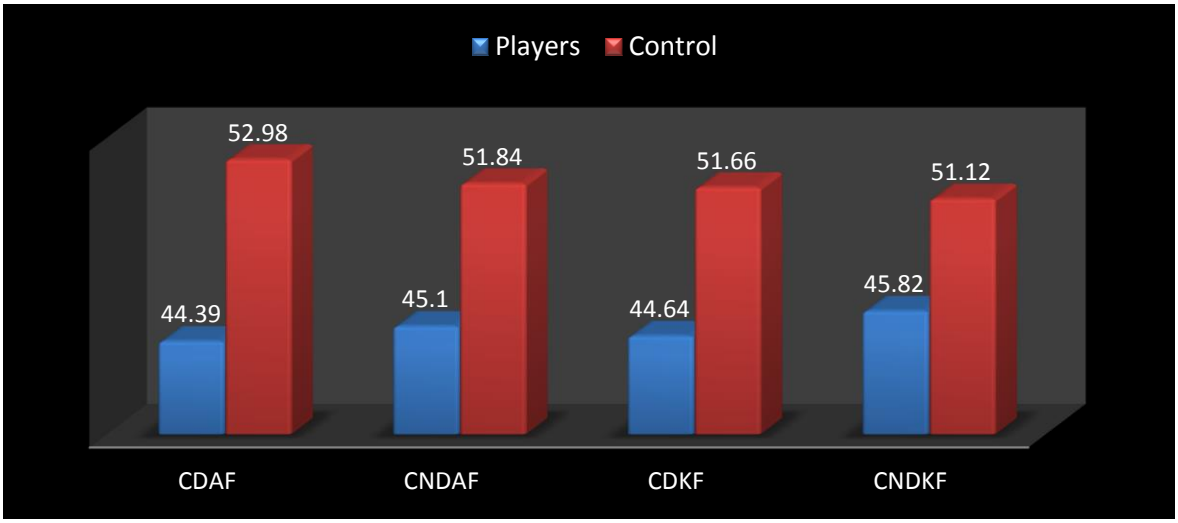


Non-Dominant Limb



In both dominant limb and non-dominant limb, the mean value of conduction velocity of common peroneal nerve of players was lower than that of controls. This shows a decreased nerve conduction in players than controls

Chart 45: F-WAVE LATENCY



In both dominant limb and non-dominant limb, the mean value of f-wave latency of common peroneal nerve of players was lower than that of controls. This shows a decreased nerve conduction in players than controls.

DISCUSSION

DISCUSSION

Football is one of the widely played sports in the world. In this sport, entire body weight is concentrated upon the lower limbs and the stress of playing for over 90 minutes takes its toll on the legs. As a result of which both physiological and pathological changes occur in lower limbs. Lower limb nerve fibres are exposed to acute and chronic mechanical injuries[62].

In this study, the nerve conduction studies of lower limb motor nerves namely tibial and common peroneal nerves was done.

There was slight delay in motor nerve conduction velocity and also delay in F wave response in the above nerves.

The possible causes for the delay in conduction velocity could be nerve entrapment[62]. Retrospective studies indicate that muscles, tendon, bones and nerves tend to adapt in response to high training loads.

Nerve entrapments in athletes can happen because of

1. Direct trauma to the specific nerve
2. Indirect trauma to the nerve which may be a fracture of a bone impinging upon the nerve.

Direct trauma to the nerve is frequently seen in cases of common peroneal nerve wherein the nerve is commonly injured at the neck of fibula where it is most superficial.

3. Nerves can also be exposed to short time high pressure or long time low pressure.

   Pressure may be because of

   a) tight thickened fascia of muscle exerting a constrictive force on the nerve.
   b) Any bony anamoly impinging on the nerve.
   c) Muscle hypertrophy compressing upon the nerve.
   d) Soft tissue swelling as in cases of ganglions surrounding the tendon sheaths compressing upon the nerve.
   e) Excessive scarring of the muscle compartments as in cases of chronic compartment syndromes[63].
   f) Orthopedic abnormality posture as in cases of Joplin's neuritis.

In the present study there is slight delay in motor nerve conduction velocities and F wave response. This is in accordance with the study conducted by Daryoush Didehdar et al in whose study there was a significant delay in motor nerve conduction velocities of tibial and common peroneal nerves. In this study motor latency of common peroneal and tibial nerves were significantly prolonged. In this study, 20 male footballers who played football continuously for 2 days a week for more than 3 years were chosen. The control group consisted of 15 non-active male subjects who did not play football and other sports which affects lower limbs. They concluded that the delay in nerve conduction velocities

may be due to the sub-clinical nerve entrapments which may occur due to the effects of excess stress the sports have on the nerves of the lower limb[62,64].

In another study done by Ozbek et al, it was found that the ulnar nerve motor conduction velocities at the above elbow to below elbow segment of volleyball players were slower than that of the normal controls. In this study 24 volleyball players were chosen and their nerve conduction parameters were compared with that of normal controls. Though they are volleyball players, this study can be brought under discussion because both sports involve enormous amount of physical activity and training which pose a serious threat to their nerves. The authors have attributed continuous use of arms leading to subclinical entrapment neuropathy was the reason behind slower ulnar nerve conduction velocities of volleyball players[65].

In another study conducted by Jazayeri Shooshtari et al it was found that in footballers subjected to ankle sprain, there was significant delay in the motor nerve conduction velocities of tibial and common peroneal nerves. There was also significantly prolonged distal nerve latencies of tibial and common peroneal nerves. In this study, 50 volunteer students aged between 19-25 years were chosen. They were divided into 3 groups. One group comprising of 20 healthy football players whose football experience exceeded 3 years. Second group had 15 footballers who had suffered ankle sprain. The last group had 15 healthy non-sports students. They concluded that harming the lower limb nerves in football may

increase the nerve latencies and hence decrease the conductivity in lower limbs[66].

In another study conducted by Colak et al, it was concluded that sub clinical nerve entrapment neuropathy occurred in upper limb nerve conduction studies of tennis players. In this study 21 male tennis players with mean age of 27.15 years were chosen. The control group consisted of 21 non-active male subjects with mean age of 26.4 years who did not participate in any kind of regular or organized sporting activity. Motor and sensory nerve conduction studies of median, ulnar and radial nerve were done. It was found out that sensory and motor conduction velocities of radial nerve were significantly delayed in dominant arms of tennis players when compared with their non-dominant arms and with normal subjects. In this study, there were no significant differences in latencies and conduction velocities of median nerve between players and controls[67].

Even though this study was done in tennis players, this can be brought under discussion because both tennis and football,

a) Are endurance sports
b) Both involve high level of physical activity
c) There is high degree of physical trauma in both sports which might cause nerve injuries.
d) Both sports induce physiological and pathological changes to muscles, tendons, soft tissue and nerves of players very much similar to each other.

In another study conducted by Colak et al, it was concluded that many of the asymptomatic runners with abnormal nerve conduction tests may represent presymptomatic or a symptomatic neuropathy similar to the type of sub clinical entrapment neuropathy. In this study, 14 asymptomatic male middle distance runners and 14 non-active subjects were chosen. Motor and sensory nerve conduction studies of medial and lateral plantar nerve, sensory nerve conduction study of sural and superficial peroneal nerves and motor nerve conduction study of common peroneal nerve were done. The nerve conduction velocities were delayed in runners compared with the control subjects. This study could be considered for discussion because football is a sport which involve a lot of running. According to statistics on an average, a footballer runs for about 7 miles per game. Some footballers may run as much as 9.5 miles per game[68].

In another study conducted by Kamen et al, it was found out that male marathoners had the slowest posterior tibial nerve conduction velocity. A group of 91 Athletes and non-athletes were chosen. The athlete group included weight lifters, swimmers, jumpers, sprinters and distance runners. NCV of weight lifters was found to be higher of the all. Weight lifting being a sport which involved less running was associated with faster posterior tibial nerve conducting velocity. Whereas marathon which involves lot of running was associated with slowest posterior tibial nerve

conducting velocity. This shows that due to heavy running, physiological and pathological changes occur in nerves resulting in slower NCVs[69].

In another study conducted by Pawlak et al, it was found that a slight trend towards lower NCV values were noted in athletes with longer duration of practicing sport. 15 field hockey players, 17 soccer players and ten tennis players were included in the study. Controls chosen were 17 healthy non active young men. NCV of ulnar and tibial nerves were assessed. It was found that no significant differences were found in nerve conduction velocities of dominant and non-dominant limbs in each studied group[70].

Ulnar nerve conduction velocity in the elbow segment was significantly lower in field hockey players. This shows that over use and stress of playing over upper limbs has resulted in slowing of nerve conduction velocities. Tibial nerve conduction velocity of non-dominant lower limb of field hockey players was higher in comparison to tennis players and control group. There was no significant correlation between body mass and NCV as well as between height of subjects and NCV in both athletes and non-athletes[70].

In the study conducted by Louis paulo Nogueira Cabral Borges et al, 3 group of athletes were compared for their median and common fibular nerve conduction velocities. 6 middle distance runners, 4 sprinters and 5 handball players were chosen. Control group consisted of 9 individuals not involved in active sports. Motor nerve conduction velocities

of Median and common fibular nerves were done. It was found that sprinters have higher nerve conduction velocities than the control group. This is contradictory to the present study which showed delay in NCVs. The authors have attributed the increase in NCV values to lower body fat percentage, better efficient integrated neuromuscular system which facilitates neurotransmission and functional overload which cause thickening of myelin sheath thus causing an increase in conduction velocities[71,72].

In the study conducted by Sandeep Singh et al. indicated that the nerve conduction velocities of drawing arm of archers was slower than bowing arm. The study was conducted at Punjabi University, Patiala (Punjab), 13 Female archers who played the sport for 5-7 years were chosen for study. Out of 13 Archers, 10 archers with mean age (20.9 $\pm$ 1.79 years) data were taken for analysis.

Nerve conduction studies of median and ulnar nerves of both upper extremities were done. The authors have attributed the slowing of nerve conduction velocities to the physical and physiological stress in game leading to sub clinical entrapment neuropathy. This is in accordance with the present study and it can be considered for discussion because co ordination of muscles for repetitive motion requires archers to possess greater muscle strength, upper body endurance and high levels of stability which poses a serious threat to the nerves[73].

In the study conducted by Wei at al in baseball players, Ulnar nerve conduction velocities of non-injured pitchers, injured pitchers and controls were done. It was found that the ulnar NCVs of injured pitchers were sub optimal in comparison with the non-injured pitchers. Eight college baseball pitchers, without elbow injury, 8 – age matched controls who did not play baseball and 8 college baseball pitchers with a history of elbow injury were chosen. This is in accordance with the present study which suggests about the possibility of nerve injuries due to repeated micro trauma which might lead onto alteration in the conduction parameters[74].

This study also showed that the ulnar NCVs of non-injured pitchers dominant arm were faster than controls and injured pitchers. This was attributed to the adaptation to trauma associated with ball throwing[74].

The present study showed that there was sub clinical nerve involvement in football players who played the sport for 3 days a week for more than five years continuously.

SUMMARY

SUMMARY

A study was conducted to assess the motor nerve conduction parameters of lower limb nerves of football players and compared with normal controls.

After obtaining institutional ethical committee approval motor nerve conduction study of tibial and common peroneal nerves were done on football players and age matched controls. Football players were chosen from nearby football clubs in and around Coimbatore. 50 Football players and 50 controls were tested for this study.

This study was done between September 2016 and March 2017.

Anthropometric measurements like height, weight, body mass index was done. Nerve conduction study was conducted at the Department of neurology, Coimbatore medical college hospital.

The data obtained from the study was analysed statistically using SPSS software version 24.

It was found out that there was a significant delay in motor nerve conduction velocities of tibial and common peroneal nerves of football players when compared with controls. There was a significant delay in F-wave component of football players when compared with controls.

It was also found out that there was no difference between dominant leg and non-dominant leg of football players. Both showed significant

amount of delay in nerve conduction velocities when compared with the controls.

It was also found that anthropometric indices like height, weight, BMI have no influence over nerve conduction parameters.

It was concluded that playing endurance sports like football for prolonged periods causes delay in nerve conduction velocities of tibial and common peroneal nerves. Nerve conduction studies can be considered as a routine diagnostic tool to assess clinical neuro conductivity in athletes who play their sport for longer periods.

CONCLUSION

CONCLUSION

The following conclusions have been derived from the present study.

It is inferred from this study that nerve conduction parameters of football players were altered when compared with the normal controls.

It is inferred that football is a sport with high contact and it causes sub-clinical neuropathies due to nerve entrapment[62].

It is well established that playing a particular sport for prolonged periods induces both physiological and pathological changes in lower limbs structures including nerves which led to the alterations in the nerve conduction parameters. Nerve entrapment is considered to be the prime factor causing sub clinical neuropathies[65].

Nerve conduction study is a simple, non-invasive procedure which should be done as a routine investigation in athletes who play their sport for prolonged periods. This will help in diagnosing sub clinical neuropathies at an early stage.

LIMITATION OF THE STUDY

1. In this study only men were included. Women were not included.

2. Nerve conduction studies were done only in motor nerves, sensory nerves were not included.

3. Had the study been done on the injured football players the results could have been more productive.

FUTURE SCOPE OF THIS STUDY

Nerve conduction study could be established as a diagnostic tool in athletes who play their sport for longer duration.

BIBLIOGRAPHY

REFERENCES

1. Elam RP. Body fat and its relationship to tibial nerve conduction velocity in a specific population. JOSPT 1987; 8:495-7

2. Victor Valderrabano, Mark Easley. Foot and Ankle Sports Orthopedics. Springer publications.pp:49-63,171-181,249-265,299-307,383-397,459-465.

3. 2013-14 NCAA Sports Medicine Handbook. Compiled by David Klossner. 24th Edition. Aug. 2013.

4. Marco Bresadola. Medicine and Science in the life of Luigi Galvani (1737-1798). Brain Research Bulletin Vol. 46, Issue 5, 15 July 1998, Pages 367-380. Elsevier Publication

5. Whittaker, E.T. (Edmund Taylor), 1873-1956 A history of the theories of aether and electricity: from the age of Descartes to the close of the nineteenth century, 1910. P.67-72.

6. David.A.Wells, A.M. The Science of Common things; A familiar explanation of the First Principles of Physical Science for Schools, families and young students. P-290 Ivison, Blakeman, Taylor & Co Publishers.

7. www.newadvent.org "Luigi Galvani". The Catholic Encyclopedia. Vol.6. New York: Robert Appleton Company, 1909.

8. www.royalsocietypublishing.org Biographical memories of Fellows of the Royal & Society: 10 Herbert Spencer Gasser, 1888-1963. 1 Nov. 1964.

9. www.revolvy.com.kenez.J(1968). "Milestones in the development of electrophysiology (Herbert-Spencer Gasser)". Orvosi hetilap. 109(32):1779-1782.

10. Merill W.Chase and Carlton C. Hunt. National Academy of sciences. Biographical Memoir. Herbert Spencer Gasser 1888-1963. National Academies Press. Washington D.C.

11. www.Nobelprize.org .The Nobelprize in physiology or Medicine 1944. Joseph Erlanger, Herbert S. Gasser.

12. Hallowell Davis. National Academy of Sciences. Biographical Memoir. Joseph Erlanger 1874-1965. National Academies press. Washington. D.C.

13. www. royalsocietypublishing.org . John Gray. Biographical Memoirs of fellows of Royal Society:35. Bryan Harold Cabot Mathews. 14 June 1906-23 July 1986. 1 Mar 1990.

14. www.ncbi.nlm.nih.gov. British Medical Journal Vol.293. 23 Aug. 1986. P- 511 "Obituary: Sir Bryan Mathews".

15. www.royalsocietypublishing.org. Sir Alan Hodgkin. Biographical Memoirs of fellows of Royal Society: 25 Edgar Douglas Adrian, Baron Adrian of Cambridge, 30 Nov. 1889 – 4 Aug. 1977. 1 Nov. 1979.

16. www.dwc.knaw.nl. Huygens Institute-Royal Netherlands Academy of Arts and Sciences (KNAW). Citation:W.Storm Van leewen, Levensbericht E.D.Adrian, in; Jaarboek, 1977, Amsterdam, PP. 188-190.

17. Frank Brink, JR. National Academy of Sciences. Biographical Memoir. Detler Wulf Bronk 1897-1975. National Academies press. Washington D.C.

18. www.sciencedirect.com Mayer RF. The Motor unit and electromyography – the legacy of Derek Denny-Brown. J Neurolsci. 2001 Aug. 15; 189(1-2): 7-11. Elsevier Publications

19. Joel A.Vilensky, Sid Gilman, Elizabeth Dunn. Derek E Denny Brown(1901-1981): His life and influence on American Neurology Journal of Medical Biography. May 1, 1998.

20. David R.Burt. National Academy of Sciences. Biographical Memoirs, Vol.87. Martin Glover larrabee 1910-2003. The National Academies Press. Washingon D.C.

21. Mohamed Kazamel, Paula Province, Mohammad Alsharabati and Shin Oh. History of Electromyography (EMG) and Nerve Conduction Studies (NCS): A tribute to the Founding Fathers (P05.259) Journal of Neurology. Feb.12, 2013 Vol. 80 No.7 Supplement P05.259.

22. Paul McCrory, Simon Bell, Chris Bradshaw: Nerve entrapments of the lower leg, Ankle and Foot in Sport: Sports Med 2002; 32(6): 371-391.

23. www.radsource.us. Leon R. Toye, M.D: MRI Web Clinic – Aug. 2012. Baxters' Nerve (First branch of the lateral plantar Nerve)Impingement

24. Venu Akuthota, Stanley A. Herring. Nerve and Vascular injuries in Sports Medicine. Springer Publications.pp-11,12,139-161,161-171.

25. Robert E. Leach, Michael B. Purnell et al, Peroneal nerve entrapment in runners. The American Journal of Sports Medicine. Mar 1, 1989; 17:287-91.

26. Raif ozden et al. Compression of Common peroneal nerve caused by an extraneural ganglion cyst mimicking intermittent claudication. J Brachial plex peripheral Nerve Inj. 2013;8:5

27. Patrick J. McMahon, Christian Lattermann, Derek Armfield, Dane K. Wukich. Current diagnosis and treatment in Sports Medicine. Lange Publications

28. Malcolm F. Mac Nicol. Peripheral Entrapment Neuropathies. Res medica J of the Royal Medical Society, New Services No. 1, 1978/79, PP 22-27.

29. Osborne MD, Rizzo TD Jr; Prevention and Treatment of ankle sprain in athletes. Sports Med. 2003; 33(15): 1145-50.

30. Fong, D et al, 2007. A Systematic review on ankle injury and ankle sprain in Sports. Sports Medicine, 37(1), PP. 73-94.

31. Zoch C, Fialka_moser V, Quittan M; Rehabilitation of Ligamentous ankle injuries: a review of recent studies. Br J. Sports Med. 2003 Aug; 37(4):291-5.

32. Drew Slimmon, Peter Brukner: Sports ankle injuries; Assessment and Management.J Australian Family Physician Vo.39, No.1/2, Jan/Feb.-2010.

33. Handbook of Neurological Sports Medicine. Concussion and other Nervous System injuries in the athlete.Chapter 18. Editors: Anthony L. Petraglia, Julian E.Bailes, Arthur L. Day. Copyright 2015.

34. James Kernohan et al, Entrapment of the Superficial Peroneal nerve: three case reports. J Bone Joint Surg Br. 1985; 67:60-1

35. Fabre T.Piton C, Andre D, et al. Peroneal nerve entrapment. J Bone Joint Surg AM 1998; 80(1):47-53.

36. Delee and Drez's Orthopedics Sports Medicine. Principles and practice. Third Edition. Saunders Elsevier Publications. 2009.

37. Tatjana Cicvara pecina et al, Tarsal tunnel syndrome in athletes: Croatian Sports Medicine Journal, Vol.26 No.1 July 2011.

38. Schol LC, Baxter DE, Neuropathies of the foot and ankle in athletes. J.Chin Sports Med. 1990 Apr; 9(2):489-509.

39. Hamza M. Alrabai et al, Mortons Neuroma in Sports. Foot and Ankle sports Orthopedics. Springer publications.

40. Peter Brukner and Karim khan. Clinical Sports Medicine. Third Edition. McGraw Hill Publications.pp-24,557,643,636-637,669.

41. Merritt GN, Subotnick SI, Median Plantar digital proper Nerve Syndrome (Joplin's neuroma) – typical presentation J footsurg.1982 Fall; 2193):166-9.

42. Linz. JC, Conti SF, Stone DA; Foot and ankle injuries; Sports injuries: Mechanism, Prevention Treatment; Fu FH, Stone DA(eds). Lippincott Williams& Wilkins, Philadelphia 2001; 1136 ff.

43. Shan SN, Miller BS, Kuhn JE; Chronic exertional compartment Syndrome. AM J Orthop. 2004 Jul; 33(7): 335-41

44. P Volcke, JH Kirby, PL Viviers, JT Viljoen. Chronic Exertional compartment Syndrome in the forearm of a rower. SAfr J SM 2014; 26(2):62-63

45. Mathew R. Bong et al. Chronic exertional compartment Syndrome: Diagnosis and Management. Hospital for joint diseases Bulletin. Vol. 62, Numbers 3 & 4. 2005. pp- 77

46. Francis G.O'Connor et al, ACSM's Sports Medicine. A comprehensive review. Wolters Kluwer/Lippincott Williams & Wilkins Publications.p-151

47. Text book of Sports Medicine. Basic Science and Clinical aspects of Sports injury and Physical activity. Editors : Michael Kjaer et al. 2003. Blackwell Publishing.pp-532-551.

48. UrgudenM, Soyuncuey, Ozdemir H, Sekban H, Akyildiz FF, Aydin AT. Arthroscopic treatment of anterolateral soft tissue impingement of the ankle: evaluation of factors affecting outcome. Arthroscopy. 2005 Mar; 2193): 317-22.

49. Mabin D, Distal nerve compression of the leg. Clinical and electrophysiological study. J. Clin Neurophysiol 1997; 27(1):9-24

50. GK.Pal. Pravathi Pal. Nerve conduction study, Chapter 34, Text book of Practical physiology, 3rd edition, P 215-219.

51. EMG and Nerve Conduction Studies in Clinical Practice. RossitzaI.Chichkova and Lara Katzin. www.practical neurology.com / January / February 2010.

52. William W. Campbell (1999). Essentials ofElectrodiagnositc Medicine, Editor. Charles W. Mitchell. Williams and Willkins. PP 43-50, 75-88, 134, 151.

53. Misra VK, Kalita J (2006), Clinical Nerve Physiology:Nerve Conduction, electromyography, evoked potentials. 2nd edition. Elsevier Publications. PP. 1-29.

54. Binnie CD, Cooper R. Mangnieree F, Osselton JW, Prior PF, Tedman BM 92004). Clinical Neurophysiology. Volume I : EMG, Nerve Conduction and Evoked Potentials. Elsevier Publications. PP:5-7, 49-54, 58, 72, 112.

55. Ross MH, Reith EL. Perineurium: evidence for contractile elements. Science, 1969; 165:604.

56. Thomas PK, OlssonY. (1984). Microscopic anatomy and function of the connective tissue components of peripheral nerve. In Peripheral Neuropathy. 2nd edition. Eds. PJ Dyck, PK Thomas, EH lambert andRBunge. WB Saunders, Philadelphia, PA. PP 97-120.

57. Sunderland S, Bradley KC. Stress-strain phenomena in human spinal nerve roots. Brain. 1961;84:120-127

58. Sunderland S. The connective tissue of peripheral nerves. Brain.1965; 88:841-854.

59. Haller FR, Low FN. The fine structure of peripheral nerve root sheath in the subarachnoid space in the rat and other laboratory animals. American Journal of Anatomy. 1971; 131:1.

60. A Textbook of Practical Physiology. CL Ghai. 8th edition. Jaypee publishers. 2013. PP 226-227.

61. Buchthal.F, Rosenfalk P. Spontaneous electrical activity of human muscle. Electroencephalography and Clinical Neurophysilogy. 1966; 20:321.

62. Daryoush Didehdar et al. Decreased Nerve Conduction Velocity in Football players. Zahedan J Res Med Sci. 2014 June; 1696):85-88.

63. Robert Salk, Thomas J.Chang. Peripheral Nerve entrapments of the foot and ankle. The Podiatry Institute. Chap. 54.

64. Didehdar D et al, Evaluation of changes in nerve conduction velocity in the lower limbs of football players. Journal of Kerman University of Medical Sciences Vol. 20, No.6, Nov-Dec. 2013.

65. Ozbek A et al, Nerve Conduction Study of Ulnar nerve in Volleyball players. Scan J Med Sci Sports 2006; 16:197-200.

66. Jazayeri-Shoostari SM, Didehdar D, Moghtaderi-Esfahani AR. Tibial and Peroneal nerve conduction Studies in ankle sprain. Electromyogr Clin Neurophysiol 2007; 47(6);301-4.

67. Colak T et al, Nerve Conduction Study of Upper extremities in tennis players. Br J Sports Med 2004; 38:632-5.

68. Colak T et al, comparison of nerve conduction velocities of lower extremities between runners and controls. J Sci Med. Sport. 2005 Dec; 8(4):403-10.

69. Kamen, G. Taylor, P. &Beehler, P.J. (1984). Ulnar and Posterior Tibial Nerve Conduction Velocity in Athletes. International Journal of Sports Medicine, 1 (5), 26-30.

70. Pawlak. M. Kaczmarek. D, Field hockey players have different values of ulnar and tibial motor nerve conduction velocity than soccer and tennis players. Archives Italiennes de Biologie, 148:365-376, 2010.

71. Luis Paulo Nogueira Cabral Borges et al, Measurement of Motor nerve conduction velocity in three different sports. Rev. Bras Med Esporte Vol.19 No.5 Sao Paulo Sept./Oct. 2013.

72. Yi-Ming Huang et al, Nerve Conduction Velocity Investigation in athletes with trained lower extremity for well controlling movement. ISBS 2005b Beijing, China PP 503-505.

73. Sandeep Singh, Sukhpreet Kaur, Study of motor nerve conduction velocities of upper extremity in the female archers. Int. J of Phy. Edu. Sports and health 2015; 196):31-33.

74. Shun-Hwa Wei, Yeu-Jeng Jong, Ya-Ju Chang, Ulnar nerve conduction velocity in injured baseball pitchers. Arch Phys Med Rehabil Vol.86, Jan. 2005.

ANNEXURES

PROFORMA

1. Name :

2. Age :

3. Address :

4. Height :

5. Weight :

6. BMI :

7. BP :

8. Your Phone No :

FOR PLAYERS:

1. Are you a football player:
2. How many hours do you play daily:
3. How many days do you play per week:
4. Are you suffering from any neuromuscular or neurological problems :
5. Are you a diabetic:
6. Are you taking drugs continuously for any chronic illness:
7. Do you suffer from malignancy :

FOR CONTROLS.

1. Do you play sports that involve legs:
2. Are you suffering from any neuromuscular or neurological problems :
3. Are you a diabetic:
4. Are you taking drugs continuously for any chronic illness:
5. Do you suffer from malignancy :

:

CONSENT FORM

I am…… S/O, Mr ……

resident of …….

a/not a football player. I have been explained about the procedures clearly. Hence I am willing to undergo the Nerve conduction study done by Dr. G. Gowthaman, a Post Graduate student of Physiology Department of Coimbatore Medical College, Coimbatore.I am aware of withdrawing myself from this study at any time. I am not forced by anyone for this testing.

Station :

Date : Signature of the individual

ஒப்புதல் படிவம்

பெயர் வயது ஆகிய நான் உடலிங்கியல் துறை கோவை மருத்துவ கல்லூரி பட்ட மேற்படிப்பு மாணவர் ஆகிய மரு.கோ. கௌதமன் அவர்கள் ஆய்வு ஆகிய "கால்பந்து வீரர்களின் கால்களில் நரம்பு கடத்தும் திறன்" பரிசோதனை செய்து கொள்ள நான் சம்மதிக்கிறேன். இந்த ஆய்வில் செய்முறை மற்றும் இது தொடர்பான அனைத்து விளக்கங்களையும் கேட்டுக்கொண்டு எனது சந்தேகங்களையும் தெளிவுபடுத்திக் கொண்டேன் என்பதையும் தெரிவித்துக் கொள்கிறேன்.

இந்த ஆய்வில் நான் உட்பட முழு மனதாக சுயசிந்தனையுடன் ஒத்துக்கொள்வதுடன் எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து விலகிட எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இந்த ஆய்வில் என்னுடைய விவரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபணை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன்.

இடம்:

தேதி: கையொப்பம் \ கைரேகை

KEY TO MASTERCHART

TYPE P-Players

TYPE C-Controls

TDAL1-Tibial nerve-dominant leg-ankle to foot segment-proximal latency

TDAL2- Tibial nerve- dominant leg- ankle to foot segment-distal latency

TDAA- Tibial nerve- dominant leg -ankle to foot segment-amplitude

TDAD- Tibial nerve- dominant leg -ankle to foot segment-distance

TDACV- Tibial nerve- dominant leg -ankle to foot segment-conduction velocity

TDAF - Tibial nerve- dominant leg -ankle to foot segment-f wave response

TDPL1- Tibial nerve- dominant leg- knee to ankle segment-proximal latency

TDPL2- Tibial nerve- dominant leg -knee to ankle segment-distal latency

TDPA- Tibial nerve- dominant leg -knee to ankle segment-amplitude

TDPD- Tibial nerve- dominant leg -knee to ankle segment-distance

TDPCV- Tibial nerve- dominant leg -knee to ankle segment-conduction velocity

TDAF- Tibial nerve- dominant leg -knee to ankle segment-f wave latency

TNDAL1- Tibial nerve-non dominant leg-ankle to foot segment-proximal latency

TNDAL2- Tibial nerve-non dominant leg-ankle to foot segment-distal latency

TNDAA- Tibial nerve-non dominant leg-ankle to foot segment-proximal latency

TNDAD- Tibial nerve-non dominant leg-ankle to foot segment-distance

TNDACV- Tibial nerve-non dominant leg-ankle to foot segment-conduction velocity

TNDAF- Tibial nerve-non dominant leg-ankle to foot segment-f wave response

TNDPL1- Tibial nerve-non dominant leg- knee to ankle segment-proximal latency

TNDPL2- Tibial nerve-non dominant leg- knee to ankle segment-distal latency

TNDPA- Tibial nerve-non dominant leg- knee to ankle segment-amplitude

TNDPD- Tibial nerve- non dominant leg- knee to ankle segment-distance

TNDPCV- Tibial nerve- non dominant leg- knee to ankle segment-conduction velocity

TNDAF- Common peroneal nerve- non dominant leg- knee to ankle segment-f wave response

CDAL1- Common peroneal nerve -dominant leg-ankle to foot segment-proximal latency

CDAL2- Common peroneal nerve - dominant leg- ankle to foot segment-distal latency

CDAA- Common peroneal nerve - dominant leg -ankle to foot segment-amplitude

CDAD- Common peroneal nerve -dominant leg -ankle to foot segment-distance

CDACV- Common peroneal nerve - dominant leg -ankle to foot segment-conduction velocity

CDAF - Common peroneal nerve - dominant leg -ankle to foot segment-f wave response

CDKL1- Common peroneal nerve - dominant leg- knee to ankle segment-proximal latency

CDKL2- Common peroneal nerve - dominant leg -knee to ankle segment-distal latency

CDKA- Common peroneal nerve - dominant leg -knee to ankle segment-amplitude

CDKD Common peroneal nerve - dominant leg -knee to ankle segment-distance

CDKCV- Common peroneal nerve - dominant leg -knee to ankle segment-conduction velocity

CDAF- Common peroneal nerve - dominant leg -knee to ankle segment-f wave latency

CNDAL1- Common peroneal nerve -non dominant leg-ankle to foot segment-proximal latency

CNDAL2- Common peroneal nerve -non dominant leg-ankle to foot segment-distal latency

CNDAA- Common peroneal nerve -non dominant leg-ankle to foot segment-proximal latency

CNDAD- Common peroneal nerve -non dominant leg-ankle to foot segment-distance

CNDACV- Common peroneal nerve -non dominant leg-ankle to foot segment-conduction velocity

CNDAF- Common peroneal nerve -non dominant leg-ankle to foot segment-f wave response

CNDKL1- Common peroneal nerve -non dominant leg- knee to ankle segment-proximal latency

CNDKL2- Common peroneal nerve -non dominant leg- knee to ankle segment-distal latency

CNDKA- Common peroneal nerve -non dominant leg- knee to ankle segment-amplitude

CNDKD- Common peroneal nerve - non dominant leg- knee to ankle segment-distance

CNDKCV- Common peroneal nerve - non dominant leg- knee to ankle segment-conduction velocity

CNDKF- Common peroneal nerve - non dominant leg- knee to ankle segment-f wave response

MASTER CHART

SNO	NAME	TYPE	AGE	SEX	HEIGH	WEIGI	BMI	TDA L1	TDA L2	TDA A	TDA D	TDA CV	TDA F	TDP L1	TDP L2	TDP A	TDP D	TDP CV	TDP F	TND AL1	TND AL2	TND AA	TND AD	TND ACV	TND AF	TND PL1	TND PL2	TND PA	TND PD	TND PCV	TND PF	CDA L1	CDA L2	CDA A	CDA D	CDA CV	CDA F	CDK L1	CDK L2	CDK A	CDK D	CDK CV	CDKF	CND AL1	CND AL2	CND AA	CND AD	CND ACV	CND AF	CND KL1	CND KL2	CND KA	CN DKD	CND KCV	CND KF
1	GOKUL	P	19	M	169	70	24.51	4.3	6.3	13	100	###	###	4.1	6.5	15	360	###	###	4.1	6.1	10	100	45.5	44.4	3.9	6.3	12	360	43.3	42.3	3.2	5.1	4	100	33.3	###	3.3	5.3	6	380	###	43.5	3.3	5.3	6	100	35.3	43.2	3.5	5.5	4	380	33.3	41.2
2	SHIHAB	P	22	M	166	60	21.77	3.3	5.1	12	100	###	###	3.1	5.3	14	360	###	###	3.5	5.3	14	100	41.6	43.7	3.3	5.1	16	360	38.5	41.7	4.1	6.6	5	100	47.3	###	4.3	6.4	7	380	###	42.2	3.9	6.4	3	100	45.3	42.2	3.7	6.2	5	380	42.5	41.2
3	DINESH	P	21	M	176	69	22.28	4.6	5.7	21	100	###	###	4.8	5.9	23	360	###	###	4.4	5.5	23	100	40.8	40.8	4.2	5.3	21	360	42.9	42.9	3.7	6.2	12	100	43.1	###	3.5	6.4	10	380	###	49.5	3.9	5.9	10	100	41.1	47.7	3.7	6.1	8	380	43.1	45.5
4	AJAY	P	24	M	173	61	20.38	4.1	6.6	19	100	###	###	3.9	6.3	21	360	###	###	4.3	6.8	17	100	41.3	42.7	4.5	6.6	19	360	39.2	44.7	4.1	5.6	10	100	41.3	###	4.3	5.4	8	380	###	45.5	4.3	5.4	12	100	43.3	41.5	4.1	5.2	10	380	45.5	43.5
5	GAUTAM	P	22	M	167	62	22.23	3.2	7.1	22	100	###	###	3.5	6.9	20	360	###	###	3.5	7.3	23	100	41.7	60.1	3.7	7.1	25	360	43.9	58.1	4.9	6.1	5	100	40.5	###	4.7	6.3	7	380	###	46.1	4.7	6.3	7	100	38.5	46.3	4.5	6.1	5	380	37.5	44.4
6	ABBAS	P	20	M	165	68	24.98	3.7	5.4	17	100	###	###	3.9	5.1	15	360	###	###	3.5	5.6	19	100	61.1	57.8	3.3	5.8	17	360	59.1	59.8	3.3	5.2	11	100	60.4	###	3.1	5.4	9	380	###	52.3	3.5	5.3	9	100	58.4	48.3	3.7	5.5	7	380	56.7	46.3
7	JOHNSON	P	25	M	175	65	21.22	4.4	5.9	19	100	###	###	4.2	6.1	21	360	###	###	4.6	5.7	17	100	53.7	43.7	4.4	5.5	19	360	55.7	45.7	4.2	6.3	9	100	50.1	###	3.9	6.1	11	380	###	43.7	3.9	6.5	11	100	48.1	43.5	3.7	6.7	13	380	50.1	45.5
8	DANIEL	P	19	M	172	63	21.30	3.4	6.1	24	100	###	###	3.6	5.9	22	360	###	###	3.6	6.3	22	100	62.3	64.6	3.8	6.1	21	360	60.3	62.6	4.7	5.5	7	100	33.4	###	4.9	5.7	9	380	###	53.7	4.5	5.7	5	100	31.4	53.7	4.7	5.5	3	380	33.5	55.7
9	VIGNESH	P	22	M	168	64	22.68	4.8	6.6	11	100	###	###	4.5	6.4	13	360	###	###	4.6	6.8	13	100	54.5	46.7	4.8	6.6	15	360	56.5	48.7	3.6	5.9	12	100	55.9	###	3.4	5.7	10	380	###	47.3	3.4	5.7	10	100	57.9	47.3	3.6	5.9	12	380	55.5	49.5
10	VIJAY	P	23	M	170	66	22.84	3.9	7.3	19	100	###	###	3.7	7.1	17	360	###	###	4.1	7.1	21	100	37.5	45.2	3.9	7.3	19	360	39.5	47.2	4.2	6.7	3	100	40.5	###	4.4	6.5	5	380	###	54.9	3.9	6.9	5	100	38.5	51.9	3.7	6.7	7	380	40.5	53.9
11	ANBARASAN	P	20	M	169	65	22.76	3.2	7.1	23	100	###	###	3.4	7.3	21	360	###	###	3.4	7.3	21	100	40.4	43.7	3.6	7.1	23	360	38.4	41.7	3.8	5.5	7	100	63.3	###	3.6	5.3	5	380	###	51.5	3.6	5.7	5	100	61.3	55.7	3.8	5.5	3	380	63.3	57.9
12	KUMARESAN	P	19	M	171	70	23.94	4.2	6.4	14	100	###	###	4.4	6.2	16	360	###	###	4.4	6.6	12	100	60.9	55.5	4.6	6.8	14	360	62.9	57.5	3.5	5.3	5	100	40.4	###	3.7	5.5	7	380	###	43.7	3.7	5.5	3	100	42.4	43.7	3.9	5.3	5	380	46.4	45.9
13	BALAJI	P	23	M	177	63	20.11	4.7	6.5	24	100	###	###	4.9	6.7	22	360	###	###	4.5	6.3	22	100	61.5	61.2	4.7	6.1	24	360	49.3	63.2	4.3	5.3	9	100	52.6	###	4.5	5.1	11	380	###	57.9	4.1	5.1	7	100	50.6	57.7	4.2	5.3	9	380	52.8	55.5
14	MADHAN	P	21	M	166	65	23.59	3.6	7.3	18	100	###	###	3.8	7.1	20	360	###	###	3.4	7.1	20	100	63.5	64.5	3.6	7.3	22	360	65.7	66.5	4.6	5.7	12	100	43.6	###	4.8	5.5	10	380	###	45.7	4.4	5.5	10	100	41.5	49.7	4.6	5.3	8	380	43.5	51.9
15	KATHIRVEL	P	19	M	168	67	23.74	3.5	6.3	16	100	###	###	3.7	6.1	14	360	###	###	3.3	6.5	18	100	50.7	44.5	3.5	6.7	16	360	48.5	46.5	4.3	6.1	10	100	33.5	###	4.5	6.3	12	380	###	45.7	4.1	6.3	12	100	35.5	41.7	4.3	6.1	10	380	33.3	43.9
16	KUMARAN	P	20	M	172	68	22.99	3.8	5.6	12	100	###	###	4.1	5.4	10	360	###	###	3.6	5.8	14	100	40.5	49.3	3.8	5.6	12	360	38.3	47.3	3.5	5.4	5	100	62.3	###	3.7	5.2	7	380	###	46.5	3.7	5.2	7	100	60.3	46.5	3.9	5.4	9	380	58.2	48.5
17	SAMUEL	P	24	M	170	69	23.88	4.5	6.2	11	100	###	###	4.7	6.5	13	360	###	###	4.3	6.4	13	100	55.5	61.9	4.5	6.2	15	360	57.5	63.9	3.3	6.2	8	100	41.5	###	3.5	6.4	6	380	###	54.7	3.1	6.3	6	100	43.5	54.7	3.3	6.1	8	380	41.5	56.9
18	JOHN	P	23	M	173	70	23.39	4.7	6.9	17	100	###	###	4.9	7.1	15	360	###	###	4.5	6.7	19	100	42.3	44.9	4.7	6.5	17	360	39.9	46.9	4.4	5.4	4	100	64.2	###	4.6	5.2	6	380	###	42.4	4.2	5.6	6	100	62.2	42.4	4.4	5.4	4	380	64.4	44.4
19	BHARATH	P	21	M	165	61	22.41	3.1	5.4	15	100	###	###	3.3	5.2	17	360	###	###	3.4	5.6	13	100	55.7	43.3	3.2	5.4	15	360	57.7	45.3	3.4	5.8	8	100	40.1	###	3.2	5.6	10	380	###	57.7	3.2	5.6	10	100	42.1	61.7	3.4	5.4	12	380	40.1	63.9
20	KARTHIK	P	26	M	169	64	22.41	3.7	6.7	21	100	###	###	3.9	6.5	23	360	###	###	3.5	6.9	19	100	60.5	62.7	3.7	6.7	17	360	58.9	64.7	4.8	6.4	11	100	57.2	###	4.6	6.2	9	380	###	55.5	4.6	6.6	9	100	55.2	55.3	4.4	6.4	7	380	57.5	57.5
21	PRAVEEN	P	25	M	167	62	22.23	4.6	6.8	23	100	###	###	4.4	6.5	21	360	###	###	4.4	7.1	21	100	40.5	43.9	4.6	6.9	23	360	42.5	45.9	3.9	6.8	6	100	40.9	###	3.7	6.6	8	380	###	43.9	4.1	6.6	8	100	42.9	43.9	4.3	6.4	6	380	40.5	46.1
22	DINESH KUMAR	P	22	M	166	63	22.86	4.4	7.2	13	100	###	###	4.2	6.9	11	360	###	###	4.6	7.3	15	100	54.7	49.5	4.8	7.1	17	360	52.7	47.5	4.4	5.3	4	100	60.9	###	4.2	5.1	6	380	###	59.8	4.2	5.5	6	100	62.9	59.8	4.4	5.3	4	380	64.9	61.9
23	SYED	P	25	M	178	64	20.20	3.4	5.9	17	100	###	###	3.6	6.1	15	360	###	###	3.6	5.7	19	100	57.7	61.1	3.4	5.9	17	360	55.5	63.1	3.5	6.8	8	100	63.9	###	3.7	6.7	6	380	###	46.7	3.7	6.6	10	100	61.9	50.9	3.9	6.4	12	380	64.5	52.9
24	SARAVANAN	P	23	M	169	67	23.46	3.7	5.4	23	100	###	###	3.9	5.2	21	360	###	###	4.1	5.6	21	100	46.5	54.3	4.3	5.4	23	360	48.5	56.3	4.5	5.5	10	100	55.5	###	4.7	5.3	8	380	###	53.3	4.7	5.7	12	100	53.5	57.5	4.5	5.5	10	380	55.5	59.5
25	ARUNKUMAR	P	24	M	165	68	24.98	4.5	5.8	11	100	###	###	4.3	6.1	13	360	###	###	4.7	5.6	13	100	44.6	45.1	4.5	5.8	15	360	46.8	47.1	4.7	6.9	6	100	47.9	###	4.9	6.7	4	380	###	42.6	4.5	6.7	8	100	45.9	46.8	4.7	6.5	10	380	47.9	44.5
26	VASANTH	P	21	M	173	69	23.05	3.1	7.3	19	100	###	###	3.3	7.5	21	360	###	###	3.4	7.1	17	100	41.6	64.7	3.2	7.3	19	360	43.6	66.7	3.6	5.3	13	100	41.8	###	3.4	5.1	11	380	###	50.1	3.8	5.5	4	100	43.8	54.3	3.6	5.3	6	380	45.8	56.5
27	PRADEEP	P	20	M	171	70	23.94	4.7	5.3	22	100	###	###	4.5	5.1	20	360	###	###	4.4	5.5	20	100	56.7	48.6	4.6	5.3	22	360	58.9	50.6	4.5	6.3	3	100	53.7	###	4.7	6.1	5	380	###	49.3	4.7	6.5	11	100	51.7	49.2	4.5	6.3	9	380	53.7	51.3
28	PARAMESWARAN	P	19	M	170	66	22.84	3.6	5.9	12	100	###	###	3.4	5.7	14	360	###	###	3.8	6.1	14	100	55.6	51.7	3.6	6.3	16	360	57.7	53.7	3.4	5.2	11	100	33.7	###	3.6	5.1	13	380	###	55.9	3.6	5.4	13	100	35.7	55.7	3.8	5.2	11	380	37.9	53.5
29	PRABHU	P	21	M	172	65	21.97	3.3	6.9	18	100	###	###	3.1	7.1	16	360	###	###	3.3	6.7	16	100	46.9	44.9	3.5	6.5	18	360	48.9	46.9	4.6	6.5	9	100	53.8	###	4.4	6.3	11	380	###	43.5	4.4	6.3	11	100	51.8	44.5	4.6	6.1	9	380	54.3	46.5
30	VIKRAM	P	23	M	165	69	25.34	3.7	5.8	20	100	###	###	3.9	5.6	18	360	###	###	3.5	6.1	22	100	45.9	45.1	3.3	5.9	24	360	47.9	47.1	3.3	5.6	5	100	43.3	###	3.1	5.8	3	380	###	56.4	3.5	5.4	3	100	45.3	56.6	3.7	5.2	5	380	47.5	58.7
31	SAIKUMAR	P	25	M	166	68	24.68	3.9	5.2	10	100	###	###	4.1	5.4	12	360	###	###	3.6	5.5	12	100	41.9	57.1	3.8	5.7	10	360	43.9	59.1	3.2	5.9	6	100	55.5	###	3.4	5.7	4	380	###	56.5	3.3	5.7	8	100	53.5	56.9	3.5	5.5	10	380	55.5	59.1
32	SRINIVASAN	P	26	M	170	70	24.22	4.3	6.8	19	100	###	###	4.1	6.6	17	360	###	###	4.1	6.6	21	100	55.8	49.9	4.3	6.8	19	360	57.8	51.9	3.7	6.6	8	100	44.7	###	3.5	6.4	10	380	###	48.1	3.5	6.4	6	100	42.7	47.9	3.7	6.2	8	380	40.5	49.9
33	SENTHIL	P	21	M	175	60	19.59	3.2	6.1	24	100	###	###	3.4	5.9	22	360	###	###	3.5	6.3	22	100	47.6	57.7	3.7	6.1	24	360	49.6	59.7	4.9	6.4	4	100	64.6	###	5.1	6.2	6	380	###	59.3	4.7	6.2	6	100	62.6	58.9	4.5	6.4	4	380	64.6	56.5
34	NANDAKUMAR	P	22	M	166	66	23.95	4.8	5.4	15	100	###	###	4.6	5.2	17	360	###	###	4.5	5.6	17	100	52.5	44.9	4.7	5.4	19	360	54.7	42.7	4.7	5.9	10	100	33.8	###	4.9	6.1	8	380	###	44.8	4.5	5.7	8	100	35.8	44.6	4.7	5.5	10	380	37.9	46.8
35	MANOJ	P	22	M	169	62	21.71	4.2	6.9	13	100	###	###	4.4	6.7	15	360	###	###	3.9	7.1	15	100	45.8	49.3	4.1	7.3	13	360	47.9	47.1	3.8	6.1	13	100	63.6	###	3.6	5.9	11	380	###	52.4	4.1	6.3	11	100	61.6	56.6	4.3	6.1	13	380	63.6	58.7
36	RAJKUMAR	P	19	M	175	67	21.88	4.6	7.1	21	100	###	###	4.8	7.3	23	360	###	###	4.4	6.9	19	100	42.1	54.4	4.2	6.7	17	360	40.1	52.2	3.2	5.7	6	100	49.5	###	3.3	5.9	8	380	###	52.7	3.4	5.5	8	100	51.7	52.9	3.2	5.3	10	380	53.9	55.1
37	VENKATESH	P	28	M	178	68	21.46	3.6	6.4	16	100	###	###	3.4	6.2	14	360	###	###	3.8	6.6	18	100	61.8	49.9	3.6	6.8	20	360	63.8	47.9	4.3	5.5	4	100	41.9	###	4.1	5.7	6	380	###	47.5	4.5	5.7	6	100	44.1	47.5	4.3	5.5	8	380	46.4	45.5
38	KARUNAKARAN	P	26	M	170	63	21.80	4.2	6.6	19	100	###	###	4.5	6.8	21	360	###	###	4.6	6.4	21	100	47.6	41.9	4.8	6.2	19	360	49.8	43.9	4.8	6.7	6	100	51.6	###	4.6	6.9	8	380	###	54.8	4.6	6.5	4	100	53.6	54.8	4.4	6.3	6	380	55.8	57.3
39	MAHENDRAN	P	30	M	169	69	24.16	4.7	5.9	23	100	###	###	4.5	6.1	21	360	###	###	4.4	5.7	21	100	41.1	52.2	4.6	5.9	23	360	43.1	50.1	4.6	6.5	7	100	33.9	###	4.8	6.7	5	380	###	45.9	4.4	5.3	5	100	36.3	45.8	4.6	5.5	7	380	38.5	47.9
40	SURENDAR	P	25	M	165	70	25.71	3.2	5.3	14	100	###	###	3.5	5.5	16	360	###	###	3.4	5.1	12	100	45.4	50.7	3.6	5.3	14	360	47.4	52.9	3.9	5.1	9	100	61.7	###	3.7	5.3	7	380	###	47.1	4.1	5.3	7	100	63.9	51.9	4.3	5.1	9	380	65.9	54.2
41	DHANASEKAR	P	23	M	168	70	24.80	7.2	8.4	6.4	100	###	###	8.4	9.6	6.8	360	###	###	7	8.8	6.8	100	29.4	78.8	8.9	9.9	7.6	360	28.2	77.6	7.4	8.3	6.5	100	23.4	###	6.9	8.8	3.4	380	###	72.2	7.4	7.6	3.6	100	23.2	72.6	7.8	7.4	4.2	380	23.5	73.1
42	VIDYAPRAKASH	P	21	M	178	75	23.67	7.6	9.8	7.6	100	###	###	8.3	11	7.6	360	###	###	7.4	9.4	7.9	100	28	78.4	8.6	11	8.4	360	26.6	77.0	8.2	8.6	7.7	100	25.6	###	8.6	8.9	3.6	380	###	73.9	8.8	8.4	3.8	100	24.4	74.2	9.2	8.8	5.4	380	25.4	74.8
43	NARAYANAN	P	19	M	168	72	25.51	7.9	10	7.8	100	###	###	8.8	9.3	8.4	360	###	###	7.8	9.8	8.3	100	30.2	80.8	9.2	9.6	8.3	360	30.6	79.8	8.4	9.2	8.9	100	22.5	###	8.8	9.7	4.9	380	###	73.5	7.2	9.6	5.2	100	23.8	74.2	9	9.4	3.8	380	18.8	78.4
44	NARENDRAN	P	20	M	170	71	24.57	9.4	8.9	6.4	100	###	###	9.4	9.6	8.4	360	###	###	9.2	9.4	6.8	100	28.4	78	9	10	9.8	360	29.8	79.8	9.6	9.5	3.2	100	19.8	###	9.4	9.2	3.2	380	###	70.6	8.4	8.8	9.6	100	21.2	71	9.4	8.8	4.2	380	22.4	72.8
45	GNANAPRAKASH	P	21	M	175	68	22.20	8.6	11	6.8	100	###	###	7.6	12	7.4	360	###	###	8.8	9.8	7.3	100	23.6	74.3	8.2	13	8.4	360	27.4	78.4	9.2	9.8	4.3	100	22.4	###	9.7	9.6	4.6	380	###	73.8	10	9.6	9.2	100	24.2	73.6	10	9.8	5.6	380	24.8	74.2
46	SUNDAR	P	24	M	175	74	24.16	7.8	11	5.7	100	###	###	11	12	6.8	360	###	###	8.3	10.4	6.4	100	29.8	79.6	11	14	7.6	360	25.2	75.0	10	9.6	4.2	100	29.4	###	11	9	4.8	380	###	78.2	12	11	5.4	100	29.4	79	9.6	9.3	6.2	380	25.4	75.2
47	VINOTH	P	23	M	176	70	22.60	7.7	9.6	6.9	100	###	###	8.4	9.8	7.9	360	###	###	7.9	9.9	7.3	100	29	80.4	9.2	11	7.4	360	31	80.2	11	15	6.4	100	28.3	###	14	14	6.8	380	###	78.1	9.8	9.4	7.6	100	28.8	79.3	9.1	9.4	5.9	380	22.6	72.9
48	KIRUBAKARAN	P	27	M	167	73	26.18	8.8	8.8	5.6	100	###	###	9	9.6	6.4	360	###	###	9.2	9.2	6.4	100	24.6	75.3	9.4	10	6.8	360	25.6	76.3	9.6	12	6.6	100	20.8	###	9.3	9.2	7.2	380	###	70.8	8.9	9.4	7.8	100	20.8	70.4	8.8	9.2	8.2	380	20.8	71.4
49	VADIVELAN	P	25	M	168	70	24.80	7.4	9.8	7.8	100	###	###	7.6	8.8	8.8	360	###	###	7.8	10	8.3	100	28	78.4	8.2	9.6	9.2	360	29	78.8	11	14	7.2	100	21.4	###	11	13	6.8	380	###	71.2	11	10	7.4	100	21.4	71	10	8.9	7.4	380	26.9	76.2
50	PRAKASH	P	20	M	171	60	20.52	7.6	9.6	6.8	100	###	###	7.8	9.5	7.4	360	###	###	7.4	10.2	8.8	100	28.2	78.4	8.8	9.2	10	360	29.2	79.4	10	5.8	3.2	100	23.6	###	3.3	11	7.2	380	###	74.8	3.3	5.6	13	100	24.6	73.8	8.8	7.9	6.5	380	25.8	76
51	AJI ANTONY	C	18	M	175	68	25.06	3.4	5.3	18	100	45	48	4.5	6.5	23	360	54	52	3.8	5.9	20	100	40	42	4.7	7.1	15	360	44	46	3.3	5.8	9.6	100	63.2	62	4.5	6.5	9.6	380	59	57.3	3.5	5.74	10.9	100	63.2	61.9	4.6	6.7	11.1	380	57.9	55.4
52	DINESH	C	18	M	174	62	20.47	3.5	5.1	20	100	39	41	4.9	7.1	22	360	57	58	3.56	5.8	19	100	47	45.6	4.6	6.9	24	360	56.2	59	3.7	5.6	11	100	48.2	51	4.1	6.9	6.6	380	52	53.6	3.7	5.26	9.8	100	56.5	58.4	4.6	6.9	10.2	380	58.8	55

53	SASIKUMAR	C	18	M	174	65	21.46	3.1	5.6	18	100	45	47	4.3	6.5	18	360	44	42	3.69	5.6	20	100	57	59.9	4.8	7.4	21	360	45.2	42	3.5	5.6	12	100	49.6	52	4.4	6.9	8.2	380	62	64.3	3.4	5.8	13	100	44.3	41.6	4.1	6.2	9.6	380	59.7	57.1
54	GOPI	C	18	M	166	58	21.04	3.9	5.9	15	100	61	64	4.6	6.9	13	360	46	48	3.12	5.63	22	100	57.9	59.3	4.4	6.9	20	360	52.3	50	3.2	5.7	9.5	100	52.3	55	4.6	6.2	5.7	380	63	65.9	3.8	5.69	6.8	100	45.6	47.8	4.7	6.4	8.6	380	60.6	58.3
55	MOHANPRASAD	C	18	M	170	64	22.14	3.5	5.2	23	100	50	47	4.5	7	24	360	59	57	3.01	5.65	25	100	58.8	55.3	4.7	6.9	22	360	56.2	54	4	5.7	7.5	100	55.5	58	4.5	6.7	10	380	48	46.3	3.2	5.42	8.9	100	59.3	57.4	4.6	7	12.8	380	55.5	53
56	DIVAKAR	C	19	M	164	58	21.56	3.7	5.4	25	100	45	41	5	7.3	22	360	56	60	3.5	5.68	24	100	59.7	57.8	5	6.2	22	360	63.2	60	3.2	5.7	8.9	100	56.5	59	5	6.8	9.1	380	47	45.3	3.5	5.21	9.1	100	56.3	59.6	4.8	6.1	11.8	380	59.4	57.3
57	ANANDH	C	19	M	162	56	21.33	3.4	5.7	10	100	59	61	4.6	7.2	24	360	46	42	3.89	5.71	20	100	60.6	63.7	4.9	7	21	360	48.2	47	3.7	5.7	11	100	61.4	61	4.6	6.7	12	380	57	59.3	3.6	5.98	5.6	100	45.6	42.4	4.4	6.3	9.6	380	63.3	61.3
58	MOHAN	C	18	M	170	60	20.76	3.8	5.1	11	100	52	48	4.6	6	20	360	59	56	3.54	5.74	19	100	63.5	61.4	4.5	7.2	18	360	49.6	45	3.2	5.3	10	100	63.2	62	4.6	6.9	12	380	58	55.4	3.6	5.12	7.8	100	58.6	56.4	4.9	6.6	12.8	380	49.1	47.3
59	DHARMESHRAJ	C	24	M	170	58	20.06	3.2	5.8	12	100	62	59	4.1	7.3	19	360	62	60	3.33	5.26	20	100	54.4	52.9	4.1	6.3	21	360	52.3	51	3.5	5.8	9.6	100	56.5	58	4.1	6.2	11	380	59	55	3.2	5.74	8.11	100	62	60.3	4.1	6.4	10.9	380	48	51
60	SURENDRAN	C	23	M	178	62	19.56	3.5	5.6	15	100	63	61	4.7	6.5	21	360	63	61	3.56	5.8	18	100	61.3	59.1	4.4	6.5	21	360	55.5	53	3.8	5.7	8.6	100	44.3	42	4.7	6.4	9.6	380	60	57.1	3.61	5.29	10.2	100	63.4	61.2	4.7	6.4	9.8	380	51	53.4
61	KOWSHIK	C	19	M	164	60	22.3	3.6	5.6	15	100	48	52	4.6	6.9	22	360	56	59	3.65	5.69	14	100	39.1	41.8	4.6	7.1	23	360	56.5	54	4	5.4	13	100	45.6	48	4.6	7	8.5	380	61	58.3	3.62	5.24	12.3	100	56.2	58.6	4.7	6.4	13	380	45	47.4
62	RISHIKUMAR	C	21	M	170	66	22.83	3.6	5.7	25	100	47	45	4.8	6.9	22	360	45	42	3.25	5.42	18	100	48	51.3	4.5	6.5	24	360	61.4	64	3.1	5.2	12	100	59.3	57	4.8	6.1	8.6	380	55	53	3.63	5.45	8.45	100	45.2	41.5	4.2	6.1	6.8	380	39	41.4
63	SATHISHKUMAR	C	18	M	174	66	21.79	3.2	5.7	16	100	57	55	4.4	6.2	24	360	52	50	3.52	5.21	13	100	51	53.5	5	6.9	20	360	63.2	61	3.5	6	9.6	100	56.3	60	4.4	6.3	9.4	380	59	57.3	3.64	5	8.56	100	52.3	49.9	4.3	6.8	8.9	380	45	47.4
64	RAVISHANKAR	C	19	M	174	68	22.46	3.6	5.7	12	100	58	55	4.7	6.7	23	360	56	55	3.14	5.98	11	100	45	47.2	4.6	7	21	360	56.5	58	3.7	5.1	13	100	45.6	42	4.9	6.6	9.7	380	63	61.3	3.65	5.42	9.35	100	56.2	54.8	4.1	6.7	9.1	380	60.9	63.3
65	RAJENDRAN	C	20	M	176	70	22.59	3.6	5.7	19	100	59	56	5	6.8	22	360	63	61	3.74	5.12	18	100	39	42.3	4.6	7.3	25	360	44.3	42	3.3	5.7	11	100	58.6	56	4.1	6.4	10	380	49	47.3	3.67	5.62	9.68	100	63.2	61.3	4.8	6.9	5.6	380	50	52.4
66	GURUPRASATH	C	20	M	168	68	24.09	3.6	5.3	14	100	60	56	4.9	6.7	24	360	48	50	3.41	5.74	15	100	45	43.2	4.1	7.2	21	360	45.6	48	3.3	5.3	9.8	100	62	60	4.7	6.4	11	380	48	51	3.68	5.98	10.3	100	48.2	49.5	4.7	6.2	7.8	380	45	42.4
67	ELAYAVEL	C	21	M	176	70	22.59	3.6	5.8	22	100	61	57	4.5	6.9	10	360	50	47	3.17	5.56	22	100	60.9	57.2	4.7	6	25	360	59.3	57	3.2	5.2	13	100	63.4	61	4.7	6.4	12	380	51	53.4	3.69	5.45	11	100	49.6	47.1	4.2	6.4	8.11	380	59	56.4
68	LOGESWARAN	C	18	M	170	65	22.49	3.7	5.7	24	100	55	58	4.1	7.3	20	360	52	50	3.71	5.57	22	100	50	47.3	4.6	7.3	23	360	56.3	60	3.3	5.5	6.8	100	56.2	59	4.2	6.1	11	380	45	47.4	3.45	5.3	12.4	100	52.3	49.8	4.5	7	10.2	380	51.6	49
69	GOKUL	C	18	M	180	64	19.75	3.7	5.4	14	100	59	58	4.4	7.2	24	360	56	52	3.8	5.58	25	100	52.1	54.7	4.8	6.5	22	360	45.6	42	4	5	8.9	100	45.2	42	4.3	6.8	9.3	380	39	41.4	3.78	5.1	11.1	100	55.5	51.8	4.3	6.1	12.3	380	62	59.4
70	INIYAN	C	18	M	180	66	20.3	3.7	5.2	16	100	63	59	4.6	6.2	22	360	57	55	3.56	5.58	23	100	56.3	58.9	4.4	6.9	18	360	58.6	56	3.8	5.4	9.1	100	52.3	50	4.1	6.7	8.9	380	45	47.4	3.65	5.6	9.3	100	56.5	54.6	4.7	6.3	10.9	380	63	60.4
71	SAGAR	C	19	M	170	70	24.22	3.7	6	18	100	49	59	4.5	7.1	24	360	61	63	3.69	5.59	25	100	48.9	51.5	4.9	6.9	13	360	62	60	3.9	5.6	5.6	100	56.2	55	4.8	6.9	9.4	380	61	63.3	3.98	5.9	8.9	100	61.4	63.4	4.3	6.6	9.5	380	48.4	45.8
72	SIDHARTH	C	19	M	168	66	23.38	3.5	5.1	20	100	48	45	5	7	16	360	54	57	3.12	5.6	24	100	49.3	51.9	4.1	6.2	24	360	63.4	61	3.9	6	7.8	100	63.2	61	4.7	6.2	7.9	380	50	52.4	3.12	5.2	9.36	100	54	57.1	4.3	6.4	8.36	380	47	44.4
73	VARUN	C	19	M	166	57	20.68	3.8	5.7	18	100	51	54	4.6	6	19	360	57	58	3.01	5.61	24	100	46.7	49.3	4.7	6.7	22	360	56.2	59	3.3	5.5	8.1	100	48.2	50	4.2	6.4	6.4	380	45	42.4	3.14	5.4	9.6	100	56.5	58.1	4.3	6.3	11.1	380	57	54.3
74	PUVIYARASAN	C	25	M	176	74	23.88	3.7	5.3	15	100	45	48	4.6	6	23	360	44	48	3.5	5.61	23	100	56.9	59.5	4.7	6.8	24	360	45.2	42	3.7	5.3	10	100	49.6	47	4.5	7	9.7	380	59	56.4	3.25	5.7	10.5	100	59.3	61.7	4.3	6.8	10.6	380	57.9	55.2
75	TAMILVEL	C	23	M	174	70	23.12	4	5.2	23	100	39	41	4.1	6.1	22	360	46	43	3.89	5.72	18	100	58.3	60.9	4.2	6.7	20	360	52.3	50	3.5	5.1	12	100	52.3	50	4.3	6.1	8.4	380	52	49	3.78	5.1	12	100	41.2	43.8	4.3	6.9	10.2	380	51.9	49.2
76	SRINIPRAKASH	C	22	M	176	75	24.21	3.1	5.5	25	100	45	47	4.7	6.7	22	360	59	62	3.54	5.83	17	100	55.6	58.2	4.3	6.9	19	360	56.2	55	3.2	5.6	11	100	55.5	52	4.7	6.3	7.4	380	62	59.4	3.15	5.8	9.5	100	49.5	51.9	4.2	6.5	12.5	380	50.7	48.1
77	GNANASEKAR	C	23	M	174	64	21.13	3.1	5	10	100	61	64	4.6	7.2	24	360	56	58	3.33	5.93	20	100	49.3	46.9	4.1	7.3	21	360	63.2	61	3.2	5.9	9.5	100	56.5	55	4.3	6.6	11	380	63	60.4	3.89	5.6	7.5	100	46.5	49.1	4.2	6.6	13	380	49.6	46.9
78	ARUNKUMAR	C	22	M	176	66	21.3	3.3	5.4	11	100	50	47	4.8	6.9	21	360	46	48	3.56	5.64	18	100	62.3	59.9	4.8	7.2	22	360	48.2	50	3.2	5.2	8.4	100	61.4	63	4.3	6.4	12	380	48	45.8	3.15	5.63	8.9	100	42.9	45.3	4.2	6.4	8.45	380	48.4	45.8
79	DHARMARAJ	C	22	M	178	64	20.19	3.8	5.6	12	100	45	41	4.4	7	20	360	59	61	3.65	5.65	14	100	62	59.6	4.7	6.2	22	360	49.6	47	3.7	5.4	11	100	54	57	4.3	6.3	11	380	47	44.4	3.65	5.65	11.1	100	43.9	46.5	4.2	6.3	8.56	380	47.3	44.6
80	SURENDRAN	C	22	M	180	78	24.07	3.1	6	15	100	59	61	4.9	7.3	22	360	62	64	3.25	5.56	18	100	60.9	58.5	4.2	7.1	24	360	52.3	50	3.5	5.7	11	100	56.5	58	4.3	6.8	9.3	380	57	54.3	3.12	5.23	10.2	100	55.3	57.7	4.2	6.3	9.35	380	46.2	43.5
81	AJAY	C	23	M	172	65	21.97	3.9	5.5	15	100	52	48	4.1	7.2	22	360	63	59	3.52	5.76	13	100	48.6	46.2	4.5	7	23	360	55.5	52	4	5.1	10	100	59.3	62	4.3	6.9	9.4	380	58	55.2	3.78	5.69	9.6	100	58.3	60.9	4.7	7	9.68	380	38.6	41.5
82	PERUMAL	C	23	M	168	66	23.38	3.2	5.3	25	100	62	59	4.7	6	21	360	56	59	3.14	5.67	11	100	59.3	56.9	4.3	6	22	360	56.5	55	3.6	5.8	13	100	41.2	44	4.2	6.5	9.1	380	52	49.2	3.45	5.14	8.6	100	49.3	51.7	5	6.3	10.3	380	39.4	41.4
83	VISHNUSHANKAR	C	23	M	174	64	21.13	3.7	5.1	16	100	63	61	4.7	7.3	18	360	45	42	3.74	5.48	18	100	41.2	38.8	4.7	6	24	360	61.4	63	3.7	5.6	13	100	49.5	52	4.2	6.6	12	380	51	48.1	3.69	5.65	12.3	100	62.3	64.9	4.7	6.4	11	380	49.3	51.3
84	KARTHIKEYAN	C	18	M	170	66	22.83	3.1	5.6	12	100	48	52	4.2	6.5	21	360	52	50	3.41	5.9	15	100	49.5	47.1	4.3	6.1	10	360	54	57	3.5	5.6	11	100	46.5	49	4.2	6.4	12	380	50	46.9	3.25	5.26	11.4	100	62	59.9	5	6.5	12.4	380	55.6	57.6
85	KALAISELVAN	C	18	M	178	73	23.04	3.8	5.9	19	100	47	45	4.3	6.9	21	360	56	54	3.17	5.87	22	100	46.5	44.1	4.3	6.7	20	360	56.5	58	3.9	5.7	12	100	42.9	45	4.2	6.3	11	380	48	45.8	3.78	5.87	13	100	60.9	58.1	4.8	7	11.1	380	59.5	61.5
86	MOHANRAJ	C	18	M	176	72	23.24	3.5	5.2	14	100	57	55	4.1	6.9	23	360	63	60	3.71	5.23	22	100	42.9	45.9	4.3	7.2	24	360	44.3	48	3.5	5.2	8.3	100	43.9	46	4.2	6.3	9.6	380	47	44.6	3.98	5.12	9.36	100	39.7	41.6	4.3	6.8	9.3	380	63.6	65.6
87	RAJADURAI	C	23	M	176	80	25.82	3.7	5.4	22	100	58	55	4.8	6.2	24	360	48	47	3.52	5.1	25	100	43.9	46.9	4.3	6.9	22	360	45.6	43	3.2	5.7	5.6	100	55.3	58	4.7	7	9.6	380	46	43.5	3.56	5.98	8.32	100	41.2	43.9	4.1	6.7	8.9	380	59	57.3
88	MANIKANDAPRABU	C	23	M	178	70	22.09	3.3	5.7	24	100	52	50	4.7	7	21	360	50	45	3.54	5.7	23	100	55.3	58.3	4.3	7	24	360	59.3	62	4	5.1	7.8	100	58.3	61	5	6.3	6.6	380	39	41.5	3.47	5.42	9.65	100	45.6	48.3	4.5	6.5	9.36	380	51.6	53.6
89	RUPESH	C	22	M	176	68	21.95	3.8	5.1	14	100	51	49	4.2	7	24	360	52	51	3.56	5.36	25	100	58.3	61.3	4.2	7.3	16	360	56.3	58	3.2	5.7	8.1	100	49.3	52	4.7	6.4	8.2	380	39	41.4	3.94	5.62	10.2	100	55.6	58.3	4.1	6.9	7.85	380	62	64.3
90	BHUVANESH	C	23	M	180	74	22.83	4	5.8	16	100	50	47	4.5	6.3	18	360	56	53	3.58	5.89	20	100	49.3	52.3	4.2	7.2	19	360	45.6	48	3.7	5.3	10	100	62.3	65	5	6.5	5.7	380	49	51.3	3.56	5.98	9.6	100	43.6	46.3	4.4	6.9	6.36	380	63	65.9
91	NANDAKUMAR	C	18	M	168	62	21.96	3.6	5.6	20	100	48	46	4.3	7	22	360	57	54	3.6	5.23	18	100	62.3	65.3	4.2	6	23	360	58.6	61	3.2	5.9	12	100	62	60	4.8	7	10	380	56	57.6	3.21	5.45	8.6	100	45.3	48	4.6	6.2	9.68	380	48.4	46.3
92	VYSAKH	C	23	M	168	60	21.25	3.5	5.6	19	100	47	45	4.7	6.6	21	360	61	64	3.62	5.18	14	100	62	58.8	4.2	7.3	22	360	62	64	3.5	5.1	11	100	60.9	58	4.3	6.8	12	380	60	61.5	3.12	5.3	12.8	100	44.6	47.3	4.5	6.7	8.35	380	47	45.3
93	BALASARATHI	C	23	M	180	70	21.6	3.9	5.7	25	100	46	44	4.3	6.4	18	360	63	61	3.64	5.62	18	100	60.9	62.8	4.2	6.5	22	360	63.4	59	3.8	6	9.6	100	39.7	42	4.1	6.7	11	380	64	65.6	3.85	5.1	11.8	100	39.5	42.2	5	6.8	7.35	380	57	59.3
94	SHANMUGAM	C	22	M	173	72	24.05	3.6	5.6	15	100	48	52	4.6	6.9	22	360	56	59	3.65	5.69	14	100	39.1	41.8	4.6	7.1	23	360	56.5	54	4	5.4	13	100	45.6	48	4.6	7	8.5	380	61	58.3	3.62	5.24	12.3	100	56.2	58.6	4.7	6.4	13	380	45	47.4
95	SASIKUMAR	C	22	M	176	74	23.88	3.6	5.7	25	100	47	45	4.8	6.9	22	360	45	42	3.25	5.42	18	100	48	51.3	4.5	6.5	24	360	61.4	64	3.1	5.2	12	100	59.3	57	4.8	6.1	8.6	380	55	53	3.63	5.45	8.45	100	45.2	41.5	4.2	6.1	6.8	380	39	41.4
96	ARUN PRAVEEN	C	23	M	176	76	24.53	3.2	5.7	16	100	57	55	4.4	6.2	24	360	52	50	3.52	5.21	13	100	51	53.5	5	6.9	20	360	63.2	61	3.5	6	9.6	100	56.3	60	4.4	6.3	9.4	380	59	57.3	3.64	5	8.56	100	52.3	49.9	4.3	6.8	8.9	380	45	47.4
97	ARUN	C	23	M	172	74	25.01	3.6	5.7	12	100	58	55	4.7	6.7	23	360	56	55	3.14	5.98	11	100	45	47.2	4.6	7	21	360	56.5	58	3.7	5.1	13	100	45.6	42	4.9	6.6	9.7	380	63	61.3	3.65	5.42	9.35	100	56.2	54.8	4.1	6.7	9.1	380	60.9	63.3
98	SUNDAR	C	23	M	176	73	23.56	3.6	5.7	19	100	59	56	5	6.8	22	360	63	61	3.74	5.12	18	100	39	42.3	4.6	7.3	25	360	44.3	42	3.3	5.7	11	100	58.6	56	4.1	6.4	10	380	49	47.3	3.67	5.62	9.68	100	63.2	61.3	4.8	6.9	5.6	380	50	52.4
99	PARTHIBAN	C	18	M	168	86	30.47	6.5	8.3	7.3	100	30	73	8.3	9.1	9.2	360	33	75	5.85	8.45	7.3	100	21.9	78.5	6.5	9.9	10	360	24.8	76	7.5	8.6	4.9	100	25.6	75	8.3	9.4	5.4	380	28	76.7	7.8	9.5	4.87	100	28.8	77.4	8.5	8.4	4.8	380	26.8	77.4
100	SABARI	C	23	M	170	84	29.06	7.6	7.9	8.4	100	31	75	7.9	9.9	9.8	360	31	78	6.24	8.35	7	100	23.8	77.2	7.1	9	9.8	360	25.6	78	7.6	8.3	5.3	100	24.8	80	9.5	9	6.7	380	26	80.4	8.4	10.8	4.89	100	24.8	75.9	8.5	10	5.4	380	29.4	78.5